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THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—14TH YEAR. SYDNEY: SATURDAY, AUGUST 13, 1927. No. 7.

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An Address.[1]

PUERPERAL SEPSIS.

By H. H. E. RUSSELL, O.B.E., V.D., M.D. (Adel.), F.R.C.P. (Edin.),

Retiring President of the South Australian Branch of the British Medical Association.

THE subject of my address this afternoon is puerperal sepsis and when choosing it I felt somewhat guilty that I was leading you over paths so often trodden during the last few years, but when we consider the outstanding importance of this subject, we must realize that as much consideration as possible must be given it by the profession generally. Furthermore as a general medical practitioner who has been actively engaged in obstetric work for over twenty-five years and also for the same length of time Health Officer for the City of Unley, I have been in touch with not only my own septic patients, but all those occurring in a community of over 42,000. I therefore thought that I could speak on puerperal sepsis from both points of view, namely that of the general practitioner and as a health officer.

We have been told frequently of late that puerperal sepsis accounts for at least one-third of maternal deaths which is between 4‰ and 5‰ and that puerperal sepsis is preventible and that as the general medical practitioner attends to most of the obstetric work in the State he is therefore the responsible person. I shall endeavour to point out that although admittedly the responsibility of the medical practitioner is great, he should not and cannot shoulder the whole blame while the unsatisfactory conditions remain as they are in this State.

According to statistics published in the official Year Book of the Commonwealth of Australia No. 19, 1925, and drawn from various countries on deaths per thousand live births, Netherlands heads the list with 0·62 and South Australia is twenty-fourth with the unenviable record of 2·36. No importance whatever can be placed on this at present, for different countries have different ways of classification and, further, sepsis comes as it were in seasons so that the very next table if the last year 1926 were taken instead of 1925, South Australia would be eighth.

If we judge results by statistics, it would be well to have a uniform mode of international classification and quinquennial periods taken. During the quinquennium 1902 to 1906 the maternal death rate for South Australia was 4·82‰ and from sepsis 1·76‰. From 1922 to 1926, twenty years later, the maternal death rate was 4·82‰ and from sepsis 1·70‰. Therefore according to the statistical tables mortality due to sepsis is just as rampant as it was twenty-five years ago and it may be thought that our efforts to combat it have not been productive of any advance during that period. I do not believe this to be the case for the following reasons:

1. Notification and classification of deaths due to puerperal causes of later years are more complete than they were.

2. Patients certified by the medical practitioner as having died from sepsis after abortion or miscarriage are now entered as deaths from puerperal sepsis. The medical practitioner is in no way to blame for these deaths. If the number of deaths from puerperal septicæmia occurring in South Australia for 1926 be taken and those cases of sepsis after abortion or miscarriage at our public hospitals be deducted, the number of deaths per thousand from puerperal septicæmia would then improve from 1·48 to 1·13 or for the last three years (the only period I could obtain statistics for) from 1·62 to 1·36.

[1] Delivered at the Annual Meeting of the South Australian Branch of the British Medical Association on June 30, 1927.

TABLE SHOWING DEATHS FROM SEPSIS PER THOUSAND LIVE BIRTHS IN SOUTH AUSTRALIA.

Year.	Births.	Maternal Deaths.	Maternal Deaths. Rate per Thousand Births.	Rate per Thousand Births for Five Years.	Deaths from Puerperal Septicæmia.	Rate per Thousand Births for Five Years.
1902	8,927	35	3·91		11	
1903	8,575	39	4·60		18	
1904	9,100	54	5·93	4·82	25	1·76
1905	8,832	34	3·85		9	
1906	9,921	52	5·83		15	
1907	9,209	46	5·00		23	
1908	9,756	45	4·61		23	
1909	10,064	50	4·97		20	2·11
1910	10,540	50	4·74		19	
1911	11,057	51	5·52		22	
1912	12,079	44	3·64		18	
1913	12,027	57	4·51		25	
1914	12,905	59	4·57		29	1·98
1915	11,798	52	4·41		19	
1916	11,857	57	5·65		31	
1917	11,326	46	4·06		16	
1918	11,357	44	3·87		12	
1919	11,060	55	4·97		17	1·42
1920	12,028	54	4·49		12	
1921	11,974	69	5·75		25	
1922	12,001	47	3·92		18	
1923	11,692	51	4·36		25	
1924	11,592	57	4·92		13	1·70
1925	11,457	75	6·55		26	
1926	11,483	50	4·35		17	

3. Another condition influencing the mortality of sepsis is the birth rate.

For South Australia during the year 1900 the population was 353,895 and the births were 9,143 and the birth rate 25·84‰, whereas in 1926 the birth rate had fallen to 20·53‰, the population being 558,883 and the births 11,483.

Today large families are rare and this naturally means the proportion of *primiparæ* to *multiparæ* has increased greatly during the last quarter of a century, with the result that in late years there are more difficult births and consequently there is more chance of sepsis.

When all these circumstances are taken into account, I think it is obvious that there has been an advance in our methods of dealing with sepsis of recent years.

This view is stated by J. Johnstone Jervis, Medical Officer of Health, Leeds, in *Public Health* of April, 1927, at page 211. After reviewing statistics dating from 1890, he sums up the position by stating:

> It would appear from the figures quoted that sepsis has responded to the efforts made to reduce the wastage of mother life better than "other causes." That at least is something to the credit of preventive medicine.

However, the improvement falls a long way short of the ideal. The figures are far too high and I am sure there are not many who are satisfied with the position as it stands today.

How can this unduly high mortality be lessened?

I believe that the solution of the problem lies in the necessity for far better practical teaching and a much more comprehensive method of notification.

Practical Teaching.

Twenty-five years ago our practical training consisted in attending twenty cases in the slums of Adelaide without any instruction from a qualified medical man. If we met a case that we considered abnormal we could summon the services of a house surgeon at the Adelaide Hospital who knew little more than we did ourselves. Was it any wonder that in our early practice our mortality from sepsis was high? My own experience during the first five years of my practice reveals a mortality per thousand from sepsis of more than twice that of the State at that time and then it gradually improved until it was much below that of the State in later years. Taken over a period of twenty-five years and involving over five thousand private patients the death rate per thousand was 1·57. Even though I allow for the adoption of better methods through experience, I am convinced that my early failure was due to a very great extent to a lack of knowledge resulting from imperfect practical instruction during my student days and it is this high mortality in early practice that can be reduced by more efficient teaching.

Present Training of the Medical Attendant.

The practical teaching of the medical student of today is a great improvement on that of former years. He is therefore able to go out into private practice far more capable than a decade ago. But even today it is grossly inadequate for efficiency. It is not long before the young practitioner realizes that the many hours he has spent at the public hospital intent on very advanced work performed by skilled specialists in other branches of his medical curriculum (work that he will probably never undertake himself unless he takes a further post-graduate course in that particular branch), could have been occupied to far greater advantage in the wider study of practical midwifery, if only the facilities were given to him to do so, but they are not. Obstetrics must be taught along similar lines to surgery and medicine. Medical students must be imbued with the idea that obstetrics is one of the most, if not the most, important and valuable of their medical duties, that fully 90% of them when qualified will be bound to attend obstetric cases and meet complications that need attention on the spur of the moment without reference to books *et cetera*. One authority states that the essentials to be aimed at in the training of the general practitioner are that he must be able to diagnose an acute abdominal condition and that he must know his midwifery. He must diagnose an acute abdominal condition. It is implied that perhaps he may obtain the services of a more skilled surgeon from a neighbouring town to perform the operation. On the other hand he must know his midwifery to combat any of the grave emergencies that may happen before he may leave the house, often under most disadvantageous circumstances in a private home and possibly supervise the anæsthetic as well.

How is it possible for a newly qualified man to have anything but a meagre knowledge of practical midwifery with four weeks' work at a maternity home where perhaps he may not have seen an abnormal case?

It is impossible for a student to spend sufficient time at a purely maternity hospital in order that he may learn his midwifery thoroughly on account of the claim other important branches of his studies have on him which necessitate his presence at the general hospital during the last three years of his course. And as so many conditions met with in midwifery are emergencies, it is often futile for the student to proceed to the maternity hospital even when notified of an abnormal case, as he usually arrives too late. It is necessary, therefore, to bring the midwifery to the student or in other words a well-equipped maternity block should be attached to the general hospital, where the student may witness deliveries daily throughout a period of two or three years, just as he is now able to do in medicine and surgery. This practice is gaining favour throughout the world and it is pleasing to note that the Adelaide Hospital is following the lead; even though it be in a very small way, it is encouraging by providing fifty midwifery beds.

Training of Nurses and Midwives.

A step in the right direction in South Australia was the training and registration of nurses and midwives.

The next generation will see the replacement of the time honoured "Gamp" by a batch of highly trained and efficient obstetric nurses. But to train nurses at the expense of the student is a mistake, for after all, is it ever necessary for a nurse to make a vaginal examination? If the condition is normal no vaginal examination is necessary and if abnormal, a vaginal examination is strongly contraindicated. If she found an abnormality would she perform version or apply forceps or deal with a *placenta prævia*? It is just as anomalous as a nurse palpating an abdomen to ascertain whether the patient is suffering from a suppurating gall bladder or an acute appendicitis; she would not operate and no good could accrue from the examination.

I agree with the professor of obstetrics who said that often the obstetric nurse with less training is allowed to do more than a trained sister in the operating theatre.

Instructing the Patient.

In recent years antenatal clinics are becoming more popular with the public generally and the expectant mother is beginning to realize the benefit of timely advice. In antenatal work we carry out: (i) Advice to the mother that she may maintain herself in better health; (ii) medical examinations to ascertain whether she is normal from a childbearing point of view and if abnormal, the corrective measures to be taken; (iii) observation and so note any danger signals which might arise, warning her of them and treating her accordingly. The ultimate aim is a healthy woman in body and mind, a normal labour and a healthy child and a mother capable of tending her children with a further reproductive life in front of her.

It has been proved that when antenatal work is carried out, instrumental interference has been lessened and resulting sepsis reduced, so that with the extension of these clinics puerperal sepsis should diminish.

Antenatal work is still in its infancy and its benefit does not reach the vast majority of women who need it. There are women who would never go to a clinic because (i) they may have some constitutional disease or complication of pregnancy which prevents them attending when most in need of attention, (ii) they have other children to look after at home and so neglect their own welfare, (iii) some do not venture outside of their own grounds during the last few weeks of pregnancy because of an inherent antipathy to being seen, (iv) others refrain from attending because of indifference.

It is not uncommon for a general practitioner to visit a home and find the expectant mother within a few days of her confinement nursing some of her offspring with an infectious fever or ministering to the needs of a septic wound. For such women it is necessary to carry out the letter of the clinic individually in their own homes by the appointment of health visitors who could be trained nurses.

These health visitors could have a health talk with the expectant mother. Their duty would be to advise as to feeding, clothing, exercise *et cetera*. The conversation between the visitor and the expectant mother should be an ordinary informal talk confined to subjects of health as applicable to the particular individual.

All literature relating to her pregnancy should be discouraged. Information should be gleaned of any existing or impending pathological condition and such reported to the practitioner who is to attend. He only should give the necessary advice for its alleviation, but the visitor should cooperate with him in seeing that his advice is carried into effect.

The medical examination includes a thorough investigation of the patient's previous health with special reference to her obstetrical history, the nature of her former confinements or miscarriages *et cetera*. The condition of her present general health and habits are ascertained and an examination of urine made. The urine is tested every fortnight from the seventh month and every week during the last month. The pelvis is examined and if thought necessary measurements are taken. A note of warning must be sounded when definite conclusions are arrived at from pelvic measurement, for adverse measurements may be the inciting factor for much unnecessary meddlesome, operative midwifery.

The size of the pelvis is certainly a factor to be considered in the management of the case, but it is by no means the only factor. The size of the fœtal head, the adaptability of the fœtal head to mould itself easily during the passage and the force of the impetus behind it are all important. Of the above unfortunately the size of the pelvis is the only one that can be ascertained with any degree of certainty and so I think too much importance can be placed on pelvic measurement.

It has been my experience and that of many others that while preparation is being made for operative treatment the baby has been born naturally because although the pelvis was contracted and malformed, the fœtal head was small, it was easily and successfully moulded and the uterine contractions were of sufficient strength to allow of a natural birth.

Educating the Public.

Before we can hope for the best results in midwifery the general public must be educated as to its importance.

The layman is only too prone to think that as for thousands of years the human offspring has made its advent into the world unaided, it will continue to do so for all time without mishap.

The onward march of civilization with all its attending evils on the human structure is ignored and so the teaching and practice of midwifery has been allowed to lag behind its sister branches, medicine and surgery, with which it is of equal, if not predominating, importance.

For this culpable neglect the populace or rather its instrument, the Government, is at fault or are we not as medical men more at fault for our neglect to force upon the public mind and make it

realize that successful delivery in a case of *placenta prævia* or such-like condition is an extremely difficult and hazardous major operation.

From the point of view of practical economy its importance cannot be overestimated, affecting as it does an otherwise perfectly healthy woman at the most important time of her life—during her reproductive period.

And so we flounder on through the mire, because we fail to make the populace shoulder its responsibility and provide sufficient moneys to cater for the efficient teaching of midwifery.

Notification and Isolation.

The *Health Act* of 1898 states among the infectious diseases to be reported puerperal fever including all puerperal conditions depending on infection.

Notification under this Act has been carried out in a very perfunctory manner and has virtually failed. The failure is attributed to several factors.

In the first place there does not exist a uniform interpretation of the Act among medical practitioners, with the result that in many instances only those cases that are likely to end fatally are reported. The Health Department quite recognizes this, but it has been difficult for it to find the cases which should have been reported and were not, because it was not notified of the birth until considerable time had elapsed. To overcome this, an *Early Notification of Births Act* became law in South Australia on December 9, 1926. Paragraph 3(1) of this Act states:

> In case of the birth of any child, the father, if actually residing in the house where the birth takes place at the time of its occurrence, or any person in attendance upon the mother at the time of, or within six hours after, the birth, shall give notice in writing of such birth to the local board of health for the district in which such birth shall take place, and such local board shall immediately report the same to the Central Board of Health.

Paragraph 3(2) states that:

> The notice shall be delivered or posted within thirty-six hours after the birth, or some longer period as determined for any district by regulation.

Paragraph 3(3) states that:

> Any local board of health shall supply without charge addressed letter cards or post cards containing the prescribed form of notice to any medical practitioner or midwife who applies for same.

This will acquaint the health authorities of every birth and permit of investigation in all abnormal cases.

In the second place because as so frequently happens, only fatal cases are reported, medical officers of health are apt to take drastic measures in all cases reported.

The coordination of the health officer with a practitioner has therefore not always been everything that could have been desired. A certain amount of antagonism between the two sometimes exists, because I think the practitioner sometimes feels that the officer of health fails to see his point of view, especially in some details which are of importance if we are to combat puerperal sepsis successfully. We are all perhaps somewhat sensitive where our midwifery is concerned, especially in a case where a temperature can occur that cannot under any circumstances be attributable to any fault on the part of the medical man in attendance.

Puerperal sepsis is described as a febrile condition of the nature of wound infection, arising during or after labour or abortion due to bacterial invasion from or absorption of products of bacterial action from the genital tract.

Any system of notification to be effective must unequivocally cover every condition arising from the above definition, the early cases as well as the late, the indefinite cases as well as the definite.

In other words the Act must be complied with. And it is my opinion that all patients with a rise of temperature to 37·8° C. (100° F.) or more on two successive days during the three weeks following the birth, whatever the cause, should be reported.

To do this without friction a more complete understanding must exist between the practitioner and health officer. A constructive, rather than a destructive policy must be their common aim, for they are both fellow workers in the field of preventive medicine. A form could be filled in by the medical practitioner in attendance on the patient, giving particulars of the confinement and puerperium and the diagnosis and if necessary the precautions taken and sent to the medical officer of health.

The medical officer of health could confer with the private practitioner with as little obtrusion as possible. If the condition proved to be one of puerperal sepsis, the health officer could take the necessary steps. These steps should not take the form of closing a hospital or maternity home, if satisfactory isolation could be carried out and every maternity home and hospital should have provision for isolation. Only in very rare cases would it be necessary to close a maternity home. Official visitations and disorganization of routine should be reduced to a minimum, consistent with the patients' welfare and the demands of public health, thereby maintaining public confidence in the practitioner and hospital.

ENDEMIC TYPHUS FEVER IN AUSTRALIA.

By FRANK S. HONE, B.A., M.B., B.S.,
Chief Quarantine Officer, South Australia; Lecturer in Clinical Medicine and in Preventive Medicine, Adelaide University.

IN three previous communications in 1921 and 1923[1][2][3] I reported a number of cases resembling typhus fever that had been observed since 1918 in Adelaide and its suburbs. On different occasions since that date and in different parts of Australia I have been asked if these cases are still occurring and questioners have been surprised to receive an affirmative reply. On this account and because similar cases have now been identified in other parts of Australia, it seems worth while to bring up to date the record of our Adelaide observations and conclusions.

Briefly it may be stated that since my last paper scarcely a month has passed without one or more of these cases being under observation somewhere in or about Adelaide. In the four years that have elapsed, we have more or less complete notes of more than eighty cases, which have been reported by more than twenty-five medical practitioners. This is nothing like a complete record, because the disease has now become such a commonplace that it often passes without special note by the medical attendant. For instance, eleven positive Weil-Felix reactions are recorded in the *Adelaide Hospital Archives* of which no clinical record has been obtained. The disease is not officially notifiable and at intervals I hear a practitioner casually mention a previous case of which no previous information has been received. Of the eighty-one cases reported in this present communication forty-seven were treated in the Adelaide Hospital. Full clinical records of seventeen of these cases can be found in the *Adelaide Hospital Archives* of 1923 and 1924, in which also are Dr. L. V. Bull's records of the results of the Weil-Felix tests. In the last two years the notes of similar cases in the Adelaide Hospital have not been published in the Archives and it does not seem worth while burdening this paper with them, as they are of the same type as those previously recorded.

Symptoms.

The cases in the present series present exactly the picture that was previously described and can be best summarized in the synopsis of symptoms and signs given in the circular which was distributed to medical practitioners in 1924.

The onset is generally sudden, with severe headache, which lasts until after the rash comes out; this is the most marked symptom in the early stages.

The eyes may be bloodshot.

Pyrexia exists from the onset, ranging between 37·8° and 39·5° C. (100° and 103° F.).

On the fifth or seventh day of illness a rash comes out, beginning like typhoid spots on the abdomen and chest; next day the spots become more numerous and spread to the shoulders and thigh and sometimes down the hands and arms.

In mild cases the rash can easily be missed; in more severe cases, when fully out, it resembles a scattered measles rash.

When fully out the rash consists of macules and papules and exists in great variation of size and intensity of spots; in severe cases some of the macules become petechial after a few days and a scattered subcutaneous discoloration is possible between the clean spots, giving the skin a dirty appearance. In severe cases the patient is often delirious in the second week and very toxic. The temperature generally comes down rather abruptly about the twelfth to fourteenth day; the rash fades about the same time or a little before. The blood does not yield a Widal reaction. The Weil-Felix reaction may be obtained during the second week, but the response to the test is often negative or doubtful late into the second week and becomes positive in dilution of over 1 in 100 by the time the temperature reaches normal.

There is generally a leucocytosis in the first week, but sometimes a leucopenia.

The condition is most likely to be mistaken for typhoid fever or measles; in mild cases the patient may not receive medical attention during pyrexia and only come under observation for a complicating pneumonia during defervescence, when careful attention to the history and the appearance of the skin will suggest the diagnosis.

The two prominent points in the clinical picture are thus the headache and the rash. The headache is more severe than in typhoid fever and is very characteristic, persisting until the rash is well developed. The rash appears from the fifth to the seventh day of the maculo-papular type previously described. It begins on the chest and abdomen and spreads over the body and extremities during the next forty-eight hours. It is generally most intense or most visible on the back and although when profuse spots can be found as far down as the hands and feet, it has a decided tendency to become more sparse as it travels down the extremities. When scanty it may be scarcely visible beyond the trunk. In only a few patients do any spots appear on the face. Definite petechiæ occur only in severe cases and in the later stages of the rash, occupying the centre of a fading macule. In addition there is the characteristic subcutaneous mottling which gives a dirty appearance to the skin. This condition combined with the variation in size and in intensity of colour of the macules, gives the appearance that I have described as a "dirty measly rash" which is characteristic of the ordinary case. On the other hand if the macules are scattered and the light not very good, a casual observer will easily mistake the macules for the rash of typhoid fever, while at the other extreme a very thick set rash will cause the subcutaneous discoloration to be overlooked and the case thought to be measles especially if the eyes are bloodshot.

Another characteristic is that on first looking at the skin a few large macules or papules are evident, which to a casual observer could easily pass for typhoid spots; continued gazing at the skin, however, makes more and more of the less defined spots become clear to view, so that after a few minutes' observation one is conscious that there is a much more profuse rash present than one thought at first. This always gives me exactly the same impression as I get when looking at the sky on a dark evening; it is not that more stars appear, but that I see more with continued gaze. In no other rash have I been so conscious of this phenomenon and it impresses me anew each time I see it.

Toxæmia varies greatly. In healthy young adults there is generally little evidence of toxæmia; once the rash has developed and the headache lessened, their general condition improves. In old people and alcoholics there is more toxæmia. In the severe

cases it becomes marked as the rash developes, so that in the second week the patient is delirious and in a typhoid state. Further experience emphasizes the difference from typhoid fever noted before, the rapid clearing up of the toxæmia at the end of the second week in patients who recover, just when in typhoid fever one would expect them to be getting worse.

Enlargement of the spleen has not been constant nor has the leucocytosis which in the earlier cases was thought to be characteristic.

Complications.

One patient under Dr. Dean Dawson in 1923 developed suppurative parotitis as the temperature was subsiding. In the previous paper two cases of jaundice in the early stages of the disease were recorded. The only abdominal complication since seen was in an old man with an umbilical hernia in whom the initial vomiting and abdominal pains were so severe as to raise the question of possible strangulation. One patient developed thrombosis of the iliac vein. The most frequent complication has been bronchopneumonia of the hypostatic type which is the usual cause of death in the fatal cases.

Course.

The majority of the patients have recovered. Of the eighty-one now recorded five died, all of them men over sixty except one woman of forty-eight with *tabes dorsalis* and cystitis. This averages about 6%, which is a little higher than in the previous series and is decidedly higher than in the series originally reported by Brill. One was a male negro, in whom the diagnosis was made only on the Weil-Felix reaction, as the rash could not be distinguished on his skin. In this patient the cause of death was bronchopneumonia. A second died suddenly of heart failure about two weeks after defervescence; a third, after defervescence, developed bronchopneumonia on which heart failure gradually supervened. Although most patients convalesce quickly, in some asthenia and anæmia persist for a long time. Experience shows that in old people especially convalescence is slow and must be carefully watched, because of the possibility of heart failure.

Diagnosis.

Diagnosis in the first week is difficult as headache and pyrexia are the main evidence of illness present. Leucocytosis varies; it is not always present in sufficiently marked degree to exclude typhoid fever.

In the second week the diagnosis rests between endemic typhus and typhoid fever or measles, according to the type of the rash. With a little care the rash can generally be differentiated and it is noteworthy that once a practitioner has had his attention directed to a case, he usually recognizes others in the next twelve months. A well-defined rash is characteristic. None the less no case should be diagnosed without a confirmatory positive Weil-Felix reaction. I had thought that I could invariably distinguish the rash from typhoid fever. However, in 1925 Dr. Cherry (who had seen a good many of these patients) sent a young girl into the Adelaide Hospital with the diagnosis of possible endemic typhus. I thought the rash was too profuse for typhoid fever and there was a history of play in rat-infested chaff sheds for a few weeks before. However, repeated Weil-Felix tests failed to elicit a reaction. The Widal test yielded a reaction. The case ran the usual course of typhoid fever and a history was also obtained of a fortnight's stay in a country district where typhoid fever was endemic. Last year after several patients with typhoid fever had been admitted to my ward from a sailing vessel in Port Adelaide and all the rest of the crew had received protective inoculation against typhoid, a month later one of them was admitted with a very profuse rash resembling endemic typhus. Subsequent progress proved beyond doubt it was typhoid fever; no reaction was obtained on any occasion to the Weil-Felix test. In this case the ordinary Widal test was impossible, because of his previous inoculation.

Dr. Lionel Bull, Director of the Pathological Laboratory at the Adelaide Hospital, has continued to test the blood of all febrile patients and has never secured a positive Weil-Felix reaction in any condition other than this disease. Now that many practitioners are becoming so used to the presence of the disease that they diagnose it on the rash and pyrexia, the importance of this confirmation by laboratory test needs to be emphasized. For instance, one boy with an almost typical rash was admitted to the Adelaide Hospital with characteristic symptoms, but for nine months prior to his coming to Adelaide the day before his illness began, he had not been away from Meningie, a country township ninety miles south of Adelaide, where no cases have yet occurred. His blood failed to give a Weil-Felix reaction throughout the pyrexial period and convalescence and his fever lasted longer than in these cases. On the other hand cases are still met with, which from their mildness or location would be overlooked, but for the positive reaction being found in the routine examination of their blood. Dr. Beare reported the case of a patient whose two daughters had had undoubted measles successively a few weeks before, in whom the rash could easily have been mistaken for measles. There were, however, no coryzal symptoms and the blood gave a definite positive Weil-Felix reaction.

The inquiries in the case of T.H. in this series (June, 1926) who had been working on a demolition job, revealed that the foreman of the same job had been attacked with similar illness ten days previously. Investigation of the hospital records showed that he was delirious and noisy when admitted, died in forty-eight hours of pneumonia, no blood examination having been made; from the *post mortem* record it is seen that death occurred from "toxæmia following an irregular pneumonia apparently of influenzal type." The whole clinical and *post mortem* picture suggests the terminal pneumonia which we have seen before in these patients and a routine Weil-Felix test would have been invaluable evidence for or against the diagnosis.

There are, however, two precautions to be observed:

1. It frequently happens that a Weil-Felix reaction cannot be obtained until defervescence has occurred. This seems more apt to occur in the mild and doubtful cases. A negative result during the pyrexial stage, therefore, does not exclude this disease and in doubtful cases the test should always be repeated after defervescence. Although most authorities speak of the reaction as occurring early in the disease, W. Fletcher and J. E. Lesslar report the same experience as ourselves in cases in the Malay States. This lessens the value of the test as an aid to diagnosis early in the disease, though enhancing its value in post-typhus pneumonia.

2. Fletcher and Lesslar[4] in writing on the Weil-Felix reaction in sporadic tropical typhus fever, point out that there are two groups of *Bacillus proteus*, one which produces indol and the other which does not. They found correspondingly two groups of cases, one which responded to the indol producing strain and one which responded only to the non-indol producing strain. The strain we have been using in Adelaide belongs to the indol producing group and it is possible that some of the cases in which we get negative results, would react to the non-indol producing strain. The reverse would be true in places where the non-indol producing train is used and this might result in cases being missed. The method of performing the test needs to be standardized throughout Australia.

Pathology.

Records of *post mortem* examinations in fatal cases can be seen in the *Adelaide Hospital Archives* for 1924. Nothing is found macroscopically beyond the ordinary appearances of septicæmia. Microscopic examination of the brain has been made twice, but the lesions said to be characteristic of epidemic typhus were not found. Examination of blood smears and of excised macules in the skin for rickettsia bodies have proved negative. Intraperitoneal injections of patients' blood into guinea pigs have invariably failed to induce the characteristic appearances. Most of these have probably been done too late in the disease, but in the case of M.B., *ætatis* seventy-one, blood was taken on the day the rash was coming out and injected intraperitoneally into two guinea pigs; no rise of temperature occurred. The guinea pigs died a month later, but nothing characteristic was seen *post mortem*.

Incubation Period.

In previous papers it was suggested that the incubation period probably averaged about fourteen days. Early support of this was secured in April, 1923, in a patient seen privately by Dr. Burston. The following are the notes of this case:

R.M., saddler, *ætatis* thirty-five, residing in North Adelaide, was seen on April 29, 1923, suffering from synovitis of the knee following on an accident at football on April 27; he was otherwise healthy. The patient was kept in bed resting the knee and was seen on May 2 and May 5, by which time the inflammation of the joint had subsided and patient was feeling quite fit. On the afternoon of May 7 he had a severe headache, which persisted for the next few days. He felt hot, but his temperature was not taken. The headache is described as coming on in spasms, almost agonizing at times; his bowels were regular. Seen on May 12, his temperature was 37·8° C. (100° F.) and his pulse rate 72. On May 14 his temperature was 39·5° C. (103° F.) in the morning and his pulse rate 80; the headaches were still very severe and spasmodic; three or four macules were noticed on his chest. By the evening the rash was thicker, about twenty spots were scattered over the chest, back and abdomen; the headache was still severe. On May 15 the rash was much thicker; the leucocyte count was 8,600. The blood agglutinated *Bacillus proteus* + 19 completely in a dilution of 1 in 160, partially in a dilution of 1 in 320 and slightly in a dilution of 1 in 640. This means a reaction. By May 20 the temperature was 37·4° C. (99·4° F.); the rash was clearing up; the headache was much easier. By May 22 the temperature was normal and from that date convalescence was uninterrupted. Another positive Weil-Felix reaction was obtained during convalescence.

It will be noticed that this man had been ten days in bed when his symptoms appeared.

In January, 1924, a lad of eighteen was admitted to the ward from premises in North Adelaide near where cases have occurred before and since. On December 24 he had gone to a country township twenty miles north of Adelaide where no cases have been known to occur, returning home on January 6, on which day his headache came on suddenly followed by the ordinary course of symptoms. He was thus just thirteen days away from possible infection.

In the accompanying chart of cases it will be noted that in several cases similar confirmatory evidence occurs. Since the *questionnaire* was put into use, it is interesting to see how often direct questions on the patients' movements about two weeks before onset of illness reveal contact with a possible source of infection.

Dissemination.

The earliest cases which were recognized in 1918 were in men who were employed in the weevilly wheat stacks in the Port Adelaide district and in my first article this association was emphasized. We still see occasional references to this point, but we have for a long time ceased to insist on this. By the time the second article had appeared, it had become evident that this association was only accidental and that it was a "place" disease rather than a purely occupational one.

An article by Megaw[5] in 1921 on a typhus-like disease in India possibly transmitted by ticks drew attention to other possibilities and gave a classification of the typhus group of diseases, differentiating groups respectively conveyed by the louse (epidemic typhus), by the tick (Rocky Mountain spotted fever) and by mites (Japanese river fever). This was referred to again by Bassett Smith in 1922. In a *post scriptum* to my second communication a case was noted (W.B.) which strengthened the idea of rats being connected with the infection.

On May 7, 1923, a man, G.C., was admitted to the Adelaide Hospital with characteristic symptoms, confirmed by the result of the Weil-Felix investigation. His home and occupational surroundings were thoroughly investigated by the Medical Officer of Health for Adelaide, the late Dr. T. Borthwick. There was nothing unusual about his home surroundings; he was employed as an assistant in a

large grocery store and for three weeks prior to the onset of his illness he had been working in the weighing room close to the packing room. Just as this report reached me I was asked by Dr. Erichsen to see a man residing on South Terrace, who had become ill on June 10 and had developed a rash. He proved to be suffering from endemic typhus and it was found that he was employed in the same store as G.C., that he had worked in the office until three weeks before the onset of his illness, when he had been transferred to the weighing room where G.C. had previously been working. Examination of the premises revealed that for a few weeks prior to this occurrence considerable alterations of the back premises adjoining the packing room had been going on and part of the building was about to be demolished. With the cooperation of the manager a thorough investigation was made by Dr. Borthwick's staff and an entomologist for the next few days. Abundant evidence of extensive rat infestation was found, extending close up to the weighing room. Through the alterations the rat population had been disturbed and odd rats could be seen. Forty-seven rats had been killed three weeks previously and six live rats and some mice were secured. Beyond this there were only the usual beetles, mealworms and the like associated with stored foods. Fleas and a mite were recovered from the captured rats, but nothing unusual was discovered in smears from the rats' blood and a Weil-Felix test on the blood of these rats gave negative results.

Reflection on these two incidents, coupled with Megaw and Smith's articles, made me think that the rodent factor was possibly more important than the food factor, as affording the possibility of a virus or an insect vector containing the virus of the disease.

From this time investigation was specially directed towards possible association of patients with rodents, such as rats or mice, which might be the host of an insect vector.

The usual paucity of cases in the latter half of the year prevented much advance for a time, but at the end of the year another odd case was notified to the State Health authorities as typhus fever or Brill's disease, with consequent confusion in statistics, seeing that there had really been eight cases in hospital that year. A conference was therefore held in January, 1924, with the President of the Central Board of Health as to whether these cases should be made notifiable for investigational purposes. He took the view that on account of its apparent slight infectionsness and probable conveyance by insect vector it was a pity to bring into operation all the clauses of the *Health Act* as regards isolation, which would be necessary if it were proclaimed a notifiable disease. He kindly consented that practitioners might continue to notify me informally of their cases, so that further investigations might be made. On this a *questionnaire* was drawn up for the Quarantine Officer to use in any case that might come under our notice. It was prefaced.

These cases are occurring sporadically, sometimes in small groups and under circumstances that suggest transmission by an insect vector, probably on a rodent, which is in some way associated with food stuffs. Inquiries should be directed especially towards these points while searching the whole field.

The results of inquiries in connexion with isolated patients admitted to the Adelaide Hospital during the next few months seemed to justify this theory of dissemination. The view was strengthened by an incident in June. Dr. J. A. Bonnin asked me to see a patient (A.J.K.) living in Hindmarsh, who worked as a tailor's cutter under good conditions in Adelaide. When I had confirmed the diagnosis, he told me that the patient's residence was two doors from a baker's shop, where in 1921 he had seen two patients, who from the subsequent articles on the subject he believed had suffered from the disease. Two days after I saw this patient he asked me to see another patient (J.) living in East Adelaide in an ideal home, who was suffering from the same complaint. Inquiries showed that he was an accountant in a butcher's shop in Hindmarsh four doors from the first patient and two doors from the baker's shop previously referred to.

Following on this a circular letter was sent in October, 1924, to medical practitioners practising in and around Adelaide summarizing the position and asking them to notify cases. No response was received for two months and it would have appeared that no notice was being taken, except for the fact that no patients with the disease were being seen in Adelaide Hospital during the same period. Suddenly in the first week in January, 1925, four cases were notified from Gilberton, Torrensville, Eastwood and North Adelaide. In January and February eleven other cases were notified. Since then cases have been more or less regularly notified, although, as stated previously, some practitioners now do not trouble. The main facts elicited from these inquiries are brought out in the accompanying table of eighty-one cases (Appendix I).

Briefly it may be stated that the inquiries have steadily tended to strengthen the positions that seemed to be established in the middle of 1923:

1. That the disease is a "place" disease;
2. That it is closely associated in some way with the prevalence of rats and mice;
3. That the influence of food stuffs is thus only indirect, by attracting and feeding these rodents;
4. That the rodent is not the sole factor, but that at times it is a harbour for infection;
5. That the infection is irregularly explosive at certain seasons and in certain localities.

To these I have gradually felt compelled to add:

6. That disturbance of material and of rodent population also plays an important part in the production of fresh cases.

If these be accepted as important factors in dissemination, it will readily be seen why our attention was first drawn to these cases in 1918 in connexion with men working amongst the weevilly wheat at Port Adelaide. Here were enormous aggregations of wheat stacks, heavily infested with mice, which in 1916 had amounted to an unprecedented mouse

plague, with an unusual amount of disturbance both of material and rat population going on, owing to the large number of men employed in shipping the stores of wheat that had accumulated during the war.

I admit the difficulty of proving these conclusions by a mere appeal to tables or charts. Where one's mind is slowly seeking for an explanation of cases that occur at irregular intervals over a few years, it is difficult to explain to others the cumulative effect of little incidents, but the evidence in favour of this being a "place" disease appears convincing. It had already been suggested by the group of five cases in Hindmarsh, recorded in a previous paper. It was strengthened by the case of W.B., occurring in February, 1923, in the same house as a previous case. In the present series it will be noted that four cases occurred at one butcher's shop in Eastwood at intervals during the early part of 1925 and that three cases occurred in thirteen months from May, 1925, to June, 1926, in another house in Hindmarsh. Taking a larger view it is striking that the majority of cases recorded in Hindmarsh have occurred in three sets of premises within a quarter of a mile of one another, that the majority of the cases recorded in Adelaide have been associated with a comparatively small residential area in the south-eastern part of the city or with a few business places outside that area. Taking a larger view still, a survey of these cases directs one mainly to the north-western, northern and eastern suburbs in addition to Adelaide itself. Remembering that the first cases occurred in Port Adelaide (which lies to the extreme north-west), one would be tempted on a superficial view to say that the infection had crept from the extreme north-west, thence up the Port Road across to the northern and north-eastern suburbs and to Adelaide. Yet there is no evidence of this and there is a considerable gap between Port Adelaide and Hindmarsh where practically no cases have occurred. Yet the freedom of the southern and western suburbs is remarkable. The cases at Henley Beach and Lockleys on the west of Adelaide all seem traceable to infection in Adelaide, as do the one or two isolated cases in Parkside and Mitcham. The only group of cases in the south-eastern suburbs is connected with a butcher's shop. The factory area in Hindmarsh is free; it is the shopping area that seems affected; but the similar shopping areas in the eastern and southern suburbs are unaffected, as are the railway goods yards in the west of Adelaide with their stores of perishable produce. Men are on the whole affected at their place of business; women, unless in positions as waitresses or shop assistants, at their residences. Yet occupation is not the primary factor, but the place of occupation. It is true that occupations connected with food stuffs still loom large in the picture, but it is the place that seems most important. In each suburb, as in Adelaide, the cases group for the most part round small loci of infection and if one could trace people's movements more thoroughly, it is probable that apparently isolated cases could be related to these foci. For instance, after six years' freedom in the Port Adelaide district two cases occur in one week in 1926 on the same premises and a fortnight later a solitary case occurs in a young girl at Alberton. Could it be found with more careful inquiry that she was related to this focus or to an Adelaide focus? Is it only a coincidence that at the end of March, 1926, a man is attacked who works at a large Rundle Street departmental store and that in the following March after the quiescent winter interval a woman gets ill, who till ten days before her onset had been for a limited period a saleswoman in the same store, and that in April another woman, living in the hills area, spends part of a day in the same store and goes down with the disease just a fortnight later?[1] It is as if in these centres reservoirs of infection exist, which at intervals overflow.

I admit the same difficulty in proving from a mere table that this reservoir lies in the rodent population. I admit the probability that if one took a series of isolated individuals and inquired about their home or occupational surroundings, one could find in a large proportion a history probably of rats and certainly of mice infesting their habitation or place of work. The evidence from isolated cases can only be subsidiary. It is of assistance when one finds in certain cases that the incubation period has been spent in a district or a ship where infection is unknown or on the other hand where, as in the case just cited, the home is in a non-infected district and a definite day about a fortnight before has been spent in an infected place. Until we know the actual cause of the infection, isolated cases cannot take us much further. But it is otherwise with the groups of cases. These are of two distinct classes whose evidence is cumulative.

First with regard to the residential groups. The only common factor in the family living in the confectioner's shop in Hindmarsh in 1922, in the undertaker's house in Hindmarsh in 1925-1926, in the house by the rubbish tip in St. Peters in 1922-1923 and in the butcher's shop in Eastwood in 1925, is the excessive infestation of the premises with rodents.

Supplementing these familial or residential groups are those groups with the same common factor: (i) living apart in healthy surroundings yet coming under the same conditions regarding rats at their place of work, for example the two grocer's assistants in Adelaide in 1923 and the two cases in which the disease reappeared in Port Adelaide, namely W.D.C. who lived at Semaphore, and J.W., living at Rosewater three miles away, yet both infected in the same week when their working premises had been so heavily infested with rats that the health authorities had been called in to deal with them; (ii) the man, A.J.K., working in clean surroundings in Adelaide and apparently infected at his rat-infested home in Hindmarsh at the same time as J. is infected at work in his butcher's shop, four doors off.

What I have termed the "explosive" character of the infection is best seen by reference to the charts. In 1923 no cases are noticed from the end of Sep-

[1] Since this article was written a man employed as a packer in the basement of the same store has suffered from the disease.

tember to the end of December, then two occur early in January, 1924. No cases are seen from July, 1924, to December 22, then three occur in twelve days. In 1925 no cases are noticed from the middle of September till December when three occur. Further, when these cases suddenly arise, they are in different suburbs with no apparent connexion with one another. The two cases in December, 1924, may have had a common focus at Walkerville, but the case early in January is in Eastwood and proves a fresh focus of subsequent infections and the case in North Adelaide at the same time is another focus. In the middle of that month a case occurs in the centre of Adelaide and at the end of the month two cases in Port Adelaide in the same building, when no case has been observed in that district since April, 1919.

What are the factors which induce these explosions? Missed mild cases that keep up the chain of infection can now be definitely excluded. If the rat or mouse is a vector, it is evident that there is no antecedent epidemic among the rodent population, as in bubonic plague, because the most careful search has brought to light no evidence of sick or dead rodents in the infested places. If the vector is a mite living on a rodent, the disease is unlike those mite-borne diseases in which the mite is an irritant, for in our cases there is a definite incubation period. It is also unlike such mite-borne diseases as Japanese river fever, for there is no rash at the original site of infection as in that disease. Regarding the ticks, there are, as far as I can ascertain, no regular tick parasites on rats in Adelaide. If an insect vector is concerned, the rat flea seems a much more likely agent. It is ubiquitous, it would account for the influence of rat disturbance, presently to be mentioned, and it would also help to account for the summer increase in cases, for it is known from the experience with bubonic plague that the flea population on rodents increases with the warm weather. Also in the warm summer of Adelaide the rat's coat becomes thinner and the flea is more apt to leave its host. But the relative paucity of cases has always been an argument against the cause lying in an insect bite alone and the sudden appearance of simultaneous cases at separated areas is rather against it being due to infected insects. All we can say at present is that the virus of the disease is in some way associated with rodents and if one could conceive of the rodent itself in some way harbouring the virus, without itself being affected with the disease, that would meet the case.

Closer study of the tables shows that the explosions are of two kinds, small limited outbreaks independent of seasons and a sudden rise in the number of cases each summer.

My mind has been increasingly impressed with the frequency with which disturbance of a hitherto quiescent rodent population has been associated with those limited explosive outbreaks which are independent of season. This was first impressed on me in the case of the two grocer's assistants in 1923, where out of a large number of employees those two men were infected who successively worked at a counter close to that part of the building the contents of which had been disturbed preparatory to demolition. I myself saw the accumulation of rubbish and rats scurrying about in the open. Reflection brought to mind that this element was present in the two previous cases connected with a rubbish tip and that it was when this element was introduced into the wheat stacks at Port Adelaide that the cases arose in sufficient numbers to force themselves on our attention. The factor would be constantly present in grocers' shops, butchering establishments, chaff sheds, auctioneers' establishments, which recur so often in the list of occupations, that one medical practitioner who has reported several cases, says that whenever he sees a case of fever in an establishment where boxes of cheese and of eggs with straw are scattered round the premises, he thinks of typhus fever. In one case, occurring in a brewery employee (W.E.S.), it was distinctly mentioned that barley which had been stored for eighteen months, was being removed with great disturbance to the population.

This factor appears also where food stuffs are not concerned, but where rat disturbance may still exist. In 1925 the caretaker of the Pirie Street Methodist Church (W.T.) became ill just after he had been working for a few weeks in the dust under flooring which had been pulled up. In the following year the demolition of the State Bank next door to this church was accompanied by the illness of the clerk of works (T.H.) and probably of the foreman. In the present year a patient in my ward, a boat proprietor (E.J.), was found to have moved recently into another house (near the above-mentioned brewery) in which the yard and outbuildings were full of rubbish; he had spent two or three days in collecting and burning this rubbish about two weeks before his illness. Two of the most recently admitted patients in the Adelaide Hospital came from two houses two hundred yards apart, which had just become infested with rats through the demolition of a chaff mill close by.

Whether such disturbances act by distributing the rodent population or by bringing it into closer contact with persons is impossible to say, but the factor is present.

This fact, coupled with the absence of any evidence of insect bites in our patients, has led Dr. L. V. Bull to suggest that the path of infection is by inhalation, which would be aided in such cases as above by the disturbance of dust. It is known that Arkwright and a collaborator while experimenting with epidemic typhus fever, inhaled the virus and contracted the disease and it is suggested that inhalation of rat excreta or of a virus associated with rat excreta may be the determining cause. The relative infrequency of cases would be in favour of a virus which is only occasionally present.

Apart from these limited outbreaks a compilation of the cases over a series of years illustrates a distinct seasonal variation, the beginning of each seasonal rise being of the explosive character referred to above.

TABLE I.—SEASONAL INCIDENCE.

Month.	1923.	1924	1925.	1926.	1927.	Total.
January ..	3	2 (1)	4	—	3	12 (1)
February ..	2	1	9	3	3	17
March	—	2	2	7 (1)	4	15 (1)
April	1	—	2 (1)	1	1	5 (1)
May	3	1	2	—	—	6
June	1	2	—	3 (1)	—	6 (1)
July	—	2	1	2	—	5
August ..	2 (1)	—	2	—	—	4 (1)
September ..	2	—	1	—	—	3
October ..	—	—	—	1	—	1
November ..	—	—	—	—	—	0
December ..	—	2	3	1	—	6
Total	14	12	26	18	11	81 (5)

The figures in parentheses are numbers of deaths.

Two possible explanations suggest themselves:

1. The effect of temperature or climatic variations. Table I leaves no doubt as to the much greater incidence of infection in the summer and autumn months, as was pointed out in the previous paper. This is in direct contradiction to epidemic typhus and coincides with the experience of those observers in America who have described similar mild outbreaks. Brill says that 70% of his cases in New York occur in the summer months and the majority of those recorded by Maxcy further south occurs in summer.

A comparison of the number of cases with the mean temperature for the summer or for individual months shows no relation between the two. In the coolest summer on record there was the same number of cases as in the preceding summer which was warmer than normal, and only one-third of those in the succeeding summer which was cooler than normal. This is shown in Tables II and III.

TABLE II.

Year.	Temperature from November to March. Above or Below Average.	Number of Cases.
1922-1923	+ 0·2°	5
1923-1924	− 3·2°	5[1]
1924-1925	− 1·5°	17
1925-1926	− 0·16°	13
1926-1927	0	11

[1] Coolest summer on record.

That temperature in itself is not a very potent factor is also suggested by the 1918 outbreak in Port Adelaide and that in Toowoomba in 1925, both of which occurred in the cooler months.

2. These two outbreaks in both of which the overwhelming number of rodents seemed to break through ordinary seasonal variations suggest that for the annual summer rise in the number of cases a sudden increase in rodent population may be a determining factor, whether or not these rodents serve as host for an insect vector. Rats in Adelaide breed throughout the winter, but there is probably an increased rate of breeding as the spring advances. The accumulated effect of spring breeding would account for the annual rise in summer and local outbreaks at other times might be due to a temporary local increase in rodent population, as well as to their distribution through outside disturbance. As previously stated, this would be still more true if the rat flea is in any way the vector, as their multiplication is greater in the warm weather. Unfortunately, here also we have no data, as no systematic study of the seasonal rise and fall in rodent population or their parasites is made by any municipality.

The advance of time has resulted in a still more powerful argument for the views here advanced. Since the *questionnaire* was first drawn up, outbreaks have occurred at Toowoomba, Port Pirie and Perth and the circumstances connected with these support our conclusions. The Toowoomba outbreak emphasizes this. The National Health Commission was in Toowoomba in May, 1925, and although I inquired personally nothing like these

TABLE III.

Months.	1922-1923.	1923-1924.		1924-1925.		1925-1926.		1926-1927.	
	Cases.	Temp.	Cases.	Temp.	Cases.	Temp.	Cases.	Temp.	Cases.
November	0	−	0	+	0	+	0	a	0
December	0	a	0	−	2	a	3	−	1
January	3	− −	2	−	4	− −	0	+	3[1]
February	2	− −	1	− −	9	+	3	− −	3
March	0	a	2	+	2	+	7	− −	4

+ = temperature above average. − = temperature below average.
a = temperature average. − − = temperature much below average.
[1] One very hot spell.

cases had been seen. In the week from June 7 to 14 seven cases are observed at different centres.[7] From June to September thirty-eight cases occurred, exactly corresponding to our clinical picture, with a positive Weil-Felix reaction, associated with a sudden large increase in mouse population and occurring almost simultaneously in separate centres of the infested districts.

In South Australia no cases had been recognized outside the suburbs of Adelaide until October last, except for a case possibly associated with Port Augusta, which is still the subject of inquiry. At Port Pirie (a seaport 130 miles from Adelaide) in October, 1926, Dr. Rees saw a patient in whom he confirmed the presence of this disease as shown by the following notes:

W.G., *ætatis* thirty-eight, a bookkeeper at an auction mart, was quite well till October 14, when he had "two sharp bites in stomach." That evening he was out of sorts, felt tired and had no appetite. Next morning he complained of anorexia and nausea on getting up. He went to work for the day, but was so ill on coming home that he had to hold on to a fence. He had an intense headache and felt hot. He continued working till October 20 with a very bad headache; he felt hot and very thirsty and was frequently in "baths of perspiration."

On October 20 he was seen by Dr. Rees. His temperature was 38·3° C. (101° F.) and his pulse rate 108. He looked ill, had a dull expression and a coated tongue and was constipated; no rash or abnormal physical signs were detected on examination.

On October 22 his temperature was 37·8° C. (100° F.). He still had a slight headache in spite of aspirin; his tongue was coated; the spleen was not palpable; there were no spots.

On October 24 he had no appetite and headache. About a dozen spots were present on the abdomen.

On October 25 his white blood cells numbered 12,000 per cubic millimetre. No organism was grown from the blood. The serum did not yield a reaction to the Widal test. The rash was more extensive.

On October 26 a maculo-papular rash appeared on the abdomen, back and chest, resembling that seen in patients at Adelaide Hospital. No *Bacilli typhosi* were isolated from the fæces and urine. There was no reaction to the Wassermann test.

On October 27 the patient's blood did not agglutinate a ten days' culture of *Bacillus proteus* × 19.

On October 28 the patient's blood agglutinated a twenty-four hours' culture of *Bacillus proteus* × 19 in a dilution of 1 in 640. The rash was almost faded.

By October 29 the patient was afebrile and had lost his headache. His temperature remained normal and he gradually improved.

The summer in Port Pirie is at least a month earlier than in Adelaide. There was the same association with a rat-infested place and the *questionnaire* was sent to Port Pirie. In February, 1927, two other cases were recognized in one of which at least there is a close association with the locality where the first patient was infected.

In Perth the first four cases were observed in December, 1926. In a private letter Dr. Dale informed me that these were in a grocer's store, with probable association with rodents. In March he wrote again that there had been six or eight more cases, two of them from the grocery store where two of the first four cases originated. This outbreak is at present under investigation by Dr. Keith Moore and it will be interesting to see how far results coincide with ours in Adelaide.

We thus have evidence of a disease occurring at several centres in Australia precisely similar in all respects to that which is endemic in Adelaide. It is interesting to speculate whether these have all spread from Adelaide and originally from Port Adelaide. This could easily be possible for Port Pirie and Perth, but it is difficult to see the connexion with Toowoomba. Moreover, this supposition could not hold and none of these suggestions as to dissemination holds with regard to the outbreak reported on the arrival of a sailing ship at Melbourne in December, 1925, by Holmes.[8]

Discussion on Nomenclature.

In the former two articles I spoke of these as a "series of cases resembling typhus fever." This was true both clinically and serologically, but it was not felt safe to go further then because the only known vector of true typhus fever was the body louse, which has been invariably absent in these cases. Since that date our knowledge has spread in two directions.

The Weil-Felix reaction has been tested out not only for the ordinary specific infections of Europe, but for dengue and other insect-borne diseases more allied to these conditions. What Maxcy wrote in 1923 has been more and more sustained: "The impression is gained that the Weil-Felix reaction is seldom, if ever, positive in normal persons nor in those diseases other than typhus which have been investigated." In the Adelaide Hospital since 1921 every patient with pyrexia of doubtful nature whose blood has been submitted to examination, has had the Weil-Felix test applied and, with two doubtful exceptions, in none but those suffering from this condition has a reaction been obtained. For this reason there now seems more ground for regarding any disease that gives a positive Weil-Felix reaction as a form of typhus fever.

On the other hand our knowledge has been extended by the recognition of other groups of mild typhus fever of which there must be some other mode of dissemination than by the body louse.

Reference has already been made to the account by Megaw in 1921 of a typhus-like disease in India. More recently, however, in 1925 it would seem that the blood of these patients does not react to the Weil-Felix test. On the other hand Fletcher and Lesslar[9] in 1925 described a series of cases in Malaya of pyrexia with but scanty rash, in which a positive Weil-Felix reaction was invariably obtained, like ours rather late in the case. The disease occurred in a limited area, in summer months, in patients who showed no evidence of body louse infection. The writers were inclined to favour ticks as vectors of the disease, but it is interesting to note that they add that there were large numbers of rats in the neighbourhood of these localities.

These rodents may be the reservoirs of the virus and harbour the larval and nymphal stages of the ticks. If this supposition is correct, the presence or absence of infected rodents will explain why the disease clings to one grazing-ground, but is absent in another which appears to have the same general characters and to support as many cattle.

TABLE SHOWING INCIDENCE OF CASES, 1923-1927.

1923.

Number and Initials.	Age.	Sex.	Address.	Occupation.	Date of Onset.	Reported by.	Probable Source of Infections.
1. A.K.	38	M.	Adelaide	Boot repairer	January 3	Dr. Shepherd	A Maltese, aged fifteen, from Port Pirie and River Murray, had been staying at house for two weeks.
2. B.McC.	15	M.	Hindmarsh	Engineering	January 29	Dr. J. H. Evans	His mother said that she suffered from same illness, having returned from Adelaide Hospital about two months before. Hospital authorities report that no reactions were obtained to tests; the diagnosis of neurasthenia was finally made. Cockroaches in house. Rats came to house from adjoining pughole.
3. W.B.	..	..	Gilberton		February 16	Dr. E. A. Brummitt	The house was close to a rubbish tip from which rats frequently infest the house. Housekeeper had same disease in August, 1922 (see Mrs. P., previous paper).
4. L.W.[1]	50	F.	Brompton		January 16		
5 L.G.[1]	17	M.	72a, West Terrace, Adelaide	Type setter	February 8		The source was not investigated.
6. H.W.[1]	17	M.	404, Gilles Street, Adelaide	Metal worker	April 9		
7. C.S.	35	M.	North Adelaide	In leather trade	May 7	Dr. S. R. Burston	Was in bed for ten days before the onset.
8. J.D.	74	M.	Adelaide	Caretaker	May 17	Dr. Duguid	
9. D.C.[1]	36	M.	51, Wakefield Street, Adelaide	Grocer's assistant	May 7	Dr. Erichsen	Both these men worked in the weighing room of a store heavily infested with rats, which was being at the time partly demolished.
10. E.P.	33	M.	Adelaide	Grocer's assistant	June 10		
11. V.W.[1]	22	F.	Argyle Street, Prospect	Shop assistant	August 17		Served in confectioner's shop in King William Street and Rundle Street, one heavily infested with rats.
12. J.G.[1] (negro)	61	M.		Builder's labourer	August 24		Died; no history obtainable.
13. A.T.[1]	40	F.	11, Devonshire Place, Adelaide	Domestic duties	September 10		Numerous rats in house in a poor part of city.
14. G.C.[1]	20	F.	Crawford Road, Hindmarsh	Waitress	September 22		

[1] Patient in hospital. In addition to above list, there were six patients in the Adelaide Hospital; according to the records the Weil-Felix reaction was obtained; probably from private practitioners, not notified.

1924.

Number and Initials.	Age.	Sex.	Address.	Occupation.	Date of Onset.	Reported by.	Probable Source of Infections.
15. E.H.[1]	18	M.	Doreen Street, Prospect	Cabinet-making	January 5		Rats at work.
16. M.B.[1]	71	M.	Gilbert Street, Adelaide.		January 11	Dr. F. S. Hone	Died suddenly of heart failure in the Adelaide Hospital when he had been convalescent a week.
17. J.S.[1]	26	M.	Parkside	In grocer's shop	February 19		
18. R.B.[1]	17	F.	Stevens Street, Adelaide		March 18		The source was not investigated.
19. F.R.[1]	56	M.	Stephen's Place, North Adelaide	Undertaker's driver	March 31	Dr. Burston	House in filthy condition; son-in-law, who lived in the same house, had similar illness a few weeks before.
20. H.B.[1]	34	M.	45, George Street, Parkside		May 3		
21. J.[1]	..	M.	St. Peters	Partner and accountant in butcher's shop	June	Dr. J. A. Bonnin	Business premises rat infested; traces of holes in cellars; another patient living four doors away was ill on June 1, 1924 (see A.J.K.).
22. A.J.K.	29	M.	Port Road, Hindmarsh	Tailor's cutter	June 1	Dr. J. A. Bonnin	Rats heard occasionally in residence.
23. R.J.D.[1]	24	M.	O'Connell Street, North Adelaide	Machinist	July 5		Rats said to be at tinsmith's next door; other cases from the neighbourhood.
24 E.W.[1]	46	F.	Adelaide	Domestic duties	July 16		Evidence of rats in residence.
25. C.W.R.	63	M.	Gilberton	Felt hatter	December 22	Dr. West	Place of occupation heavily rat infested.
26. B.A.B.	46	M.	Torrensville	Traveller for chaff and grain merchant	December 25	Dr. Beare	Rats in stable near residence. Two weeks before onset was occupied in Gilberton, where C.W.R. originated about same day.

[1] Patient in Adelaide Hospital. In addition to above list, there were five patients in Adelaide Hospital; according to the records a Weil-Felix reaction was obtained; probably from private practitioners, not notified.

TABLE SHOWING INCIDENCE OF CASES, 1923-1927—*Continued.*

1925.

[1] Patient in Adelaide Hospital. In addition to above list, there were five patients in Adelaide Hospital; according to the records a Weil-Felix reaction was obtained; probably from private practitioners, not notified.

TABLE SHOWING INCIDENCE OF CASES, 1923-1927—*Continued.*
1925.

Number and Initials.	Age.	Sex.	Address.	Occupation.	Date of Onset.	Reported by.	Probable Source of Infections.
27. E.A.P.	56	M.	Eastwood	Butcher	January 3	Dr. Arnold	Rats in and under house and shop adjoining (see also W.P., a brother, and A.C.P. and B.P., daughters of E.A.P., shown below).
28. W.E.S.	25	M.	North Adelaide	Clerk in brewery	January 6	Dr. E. A. Brummitt	Place of employment heavily rat infested; barley stored for eighteen months was being removed with great disturbance of rat population.
29. W.T.	64	M.	Pirie Street, Adelaide	Caretaker of church	January 17	Dr. Evans	Few rats seen in residence, but not in church next door. Floor of church pulled up and replaced at intervals about a week or two prior to his illness; working under floor (see T.H., 1926, State bank, next door).
30. W.D.C.	63	M.	Semaphore	Customs agent	January 30	Dr. Cherry	Place of employment heavily rat infested (see also next patient, J.W.E.).
31. J.W.E.	..	M.	Rosewater	Customs agent	February 6		Place of employment heavily rat infested. Rats stated to come from flour mill adjacent to rear of premises. Worked in same office and factory. Same desk as patient W.D.C., who was ill one week previously. Both in hospital together.
32. A.C.P.	4½	F.	Glen Osmond Road, Eastwood	At home	February 11	Dr. Arnold	Daughters of E.A.P. above. Father had disease five weeks previously. Continuously at home until two days before onset, when they began school attendance.
33. B.P.	6	F.	Glen Osmond Road, Eastwood	School	February 11	Dr. Arnold	
34. W.D.P.	71	M.	Stephen Terrace, Walkerville	Commercial agent (no connexion with foodstuffs)	February 12	Dr. Arnold	Rats seen in place of employment.
35. R.W.	6	M.	Queen Street, Alberton	School	February 19	Dr. Poole	Rats rarely seen in residence; commenced school attendance ten days before.
36. J.F.[1]	48	M.	Off Halifax Street, Adelaide	Nurseryman	February 20	Dr. Evans	No investigation.
37. A.R.[1]	32	M.	29, Hamley Street, Adelaide	Painter at motor body works	February 22	Dr. Close	No investigation.
38. C.H.[1]	17	M.	Mile End	Labourer	February 23	Emergency	No investigation.
39. W.P.[1]	54	M.	Frewville	Labourer (butcher)	February 28	Dr. Ray	Brother had similar complaint earlier in year (see E.A.P. above).
40. R.G.[1]	14	M.	15, Ballara Street, Mile End	Salesman	March 15	Dr. Griffiths	No investigation.
41. C.H.[1]	63	F.	Crowther Street, Adelaide	Domestic duties	March 18	Dr. Dawkins	
42. J.H.[1]	67	M.	Maylands	Butcher	April 7	Borthwick	Died of hypostatic pneumonia and cardiac failure. No *post mortem* examination.
43. W.J.[1]	48	M.	North Adelaide	Works in chaff store in St. Peters	April 9	Dr. Robertson	Place of employment heavily rat infested.
44. J.F.[1]	26	M.	219, Rundle Street, Adelaide	Brass polisher	May 1	Dr. Ray	Lives with father who keeps a bird-fancier's shop. Rats in residence, also in place of employment.
45. K.E.[1]	17	M.	Hindmarsh	Cabinet-making	May 22	Dr. Dolling	Rats in house; said to come from railway yards (see also R.E. below, mother, and F.E., father, in 1926 return).
46. G.	44	M.	Torrensville	Works in dye works in Gawler Place, Adelaide	July 1	Dr. F. H. Beare	Associated with children in house with measles. Rats in great profusion at wholesale dairy produce merchant across lane from place of employment.
47. J.R.[1]	34	M.	Austral Place, Norwood	Porter	August 15	Dr. Wall	No sign of rats.
48. R.E.	49	F.	Hindmarsh	Household duties	August 31	Dr. Dolling	At Point Noarlunga ten days prior to illness. Rats in great numbers at residence, said to come from adjacent railway yards. Son ill with same complaint about three months ago (see K.E. above and F.E. in 1926).
49. W.T.B.	48	M.	Kent Town	Baker	September 15	Dr. Beare	Mice on premises; rats rare; lives in his business premises, where flour is stored.
50. L.P.[1]	20	M.	Franklin Street, Adelaide	Labourer	December 8	Dr. Welch	Associated with another patient (see M.R. below); not at work for three weeks before onset; living at home.
51. M.R.[1]	19	F.	Stephen Street, Adelaide	Milliner	December 13	Dr. Letcher	Associated with L.P. above.
52. F.W.[1]	20	M.	Brownhill Creek	Packer	December 29	Dr. Gault	No investigation.

[1] Patient in Adelaide Hospital.

TABLE SHOWING INCIDENCE OF CASES, 1923-1927—*Continued.*

1926.

Number and Initials.	Age.	Sex.	Address.	Occupation.	Date of Onset.	Reported by.	Probable Source of Infections.
53. E.G.[1]	49	F.	Evandale	Household duties	February 13	Dr. Borthwick	No investigation.
54. A.J.[1]	32	M.	Fullarton Road, Parkside	Driver	February 19	A.H.	No investigation.
55. A.B.M.	38	M.	Mitcham	Salesman (seeds, grain and fodder)	February 28	Dr. K. Gault	Works at wholesale seedsmen, East End market; rats in abundance at place of employment.
56. E.L.	48	F.	North Adelaide	Household duties	March 19	Dr. F. S. Hone	Residence heavily rat infested. Also had *tabes dorsalis*; not away from home for two weeks before onset; died.
57. E.A.	35	F.	Carrington Street, Adelaide	Household duties	March 21	Dr. Beare	
58. H.L.[1]	35	M.	Port Augusta	Motor mechanic	March 23	Dr. Lendon	Rats in garage; typhoid fever prevalent at Port Augusta.
59. E.A.S.W.	..	M.	South Street, Southwark	Carter	March 25	Dr. Pitcher	Carting until onset of illness from Port Adelaide to Adelaide; went to soap factory at Hindmarsh and timber mills in Adelaide. No rats at residence.
60. A.H.	4	F.	Kent Terrace, Kent Town		March 25	Dr. Beare	Living in Kate Street, Adelaide, and five days after moving to Kent Town became ill. No rats in house at Kent Town, but there were plenty at previous residence.
61. A.S.[1]	12	M.	First Street, Brompton	Attended Hindmarsh School	March 26	Dr. Lewis	Big pughole full of rats at back of house.
62. C.B.[1]	26	M.	Seaview Road, Henley Beach	Counter-hand in department store, Rundle Street	March 29	Dr. Burden	Numerous rats at place of employment; none at house (see M.H. and W.E.P., 1927).
63. M.H.	65	M.	Stanley Street, North Adelaide	Clerk, G.P.O.	April 25	Dr. J. E. Goode	Died; no investigation.
64. T.H.[1]	47	M.	Parkside	Clerk of works, State bank	June 7	Dr. Fry	Old State bank demolished; rats probably present. House is next door but one to a grocery shop. States that a foreman on same job was seized with similar illness about ten days previously and died in Adelaide Hospital on June 1, 1926, from pneumonia. No blood examination was made (see W.T., 1925, who was caretaker next door).
65. F.E.	..	M.	Hindmarsh	Undertaker	June 7	Dr. C. E. Dolling	(See also R.E. and K.E. in 1925.)
66. J.H.[1]	43	M.	Percy Street, Prospect	Boilermaker	June 11	Dr. Goode	Works at abattoirs; rats at place of employment.
67. G.G.[1]	35	M.	Gladstone Road, Chicago	Woodworking machinist	July 4	Dr. West	Lives next to boiling down works which are heavily infested with rats.
68. A.P.[1]	44	M.	18, Eden Street, Adelaide	Labourer	July 13	Dr. Welch	No investigation.
69. C.W.B.	49	M.	Unley	Grocer	December 6	Dr. Messent	Rats in place of employment; storeman absent ill one week previously, thought to be rheumatism or sciatica.

[1] Patient in Adelaide Hosptial.

1927.

Number and Initials.	Age.	Sex.	Address.	Occupation.	Date of Onset.	Reported by.	Probable Source of Infections.
70. V.N.[1]	27	M.	Carrington Street	Salesman	January 2	Adelaide Hospital	
71. M.B.[1]	16	F.	Trinity Court, City		January 2	Adelaide Hospital	Broncho-pneumonia.
72. E.J.[1]	47	M.	Finnis Street, Lower North Adelaide	Boat proprietor	January 15	Dr. F. S. Hone	Business premises free of rats; moved to present house some weeks previously; very dirty and untidy yard. Two weeks before onset spent two or three days tidying up and burning rubbish
73. J.G.[1]	34	F.	Nottingham Street, Glenrose	Domestic duties	February 21	Adelaide Hospital	No investigation.
74. M.H.[1]	24	F.	Lockleys	Saleswoman	March 7	Dr. de Crespigny	Left Adelaide on boat trip to Port Lincoln in February, 1926; onset while on boat nine days later. Up to 26th working daily at same drapery establishment as C.B. (March 29, 1926).

[1] Patient in Adelaide Hosptial.

TABLE SHOWING INCIDENCE OF CASES, 1923-1927—*Continued.*

[1] Patient in Adelaide Hosptial.

TABLE SHOWING INCIDENCE OF CASES, 1923-1927—*Continued.*
1927—*Continued.*

Initials. Number and	Age.	Sex.	Address.	Occupation.	Date of Onset.	Reported by.	Probable Source of Infections.
75. R.D.[1]	44	M.	North Kensington	Tram conductor	March 19	Dr. de Crespigny	Numerous rats in house, which was being renovated in last three weeks. Large numbers of rats noticed about house after demolition of a chaff-house in neighbourhood; groceries were obtained from F.T. below, a grocer, about two hundred yards away.
76. M.G.[1]	68	F.	Richard Street, Hindmarsh	Household duties	March 27	Dr. Drew	Broncho-pneumonia; rats in house; large timber yards next door.
77. F.T.	55	M.	Norwood	Grocer and boot-maker	March 31	Dr. de Crespigny	House, shop and yard rat infested. Works sometimes in shed at back of house, which contains bags of wheat and barley and is badly infested with rats (see R.D. above, about two hundred yards away).
78. W.E.P.	43	F.	Blackwood	In draper's shop	April 17		In Adelaide two weeks previously ordering goods in warehouses, including drapery establishments concerned with C.B. (March 29, 1926) and M.H.

[1] Patient in Adelaide Hosptial.

PORT PIRIE.

Number and Initials.	Age.	Sex.	Address.	Occupation.	Date of Onset.	Reported by.	Probable Source of Infections.
					1926.		
79. W.G.	38	M.	Port Pirie	Book-keeper at auction mart	October 14		(See A.L. in 1927).
					1927.		
80. A.L.	..	M.	Port Pirie West	Painter in father's shop	February 4	Dr. C. Yeatman	Rats in shop; shop is in Ellen Street, next to grain store about one hundred yards away from offices and stores where illness originated in October, 1926 (see W.G. in 1926).
81. L.A.	23	M.	Port Pirie West	Labourer at smelters; assists father in fruit shop in Florence Street in spart time	February 11	Dr. C. Yeatman	Rats in one place of occupation (fruit shop).

The writers call their form tropical typhus because they think these mild cases occur more frequently in the tropics than elsewhere. It is doubtful, however, whether this really is the case.

Brill, of course, in 1910 was the first to draw attention to a series of mild cases resembling typhus fever in New York observed over a period of fourteen years. These almost exactly resembled the cases we have been seeing in Adelaide and Australia; hence the way in which the term Brill's disease has been applied to our cases by many observers. But Brill's cases were proved experimentally to be typhus fever before our cases were noticed.

In his revised article in Nelson's Loose Leaf Medicine, Brill himself describes his cases as "endemic typhus fever" as opposed to the European epidemic form.(11)

> In Boston, New York and Philadelphia cases of the endemic form are constantly occurring. For the past twenty years the writer has personally seen an average of thirty-four patients yearly with this form of the disease. . . . While the cold winter months show the greatest number of cases of the epidemic form, it is remarkable that the reverse is true of the endemic form. Seventy per cent. of all cases of the endemic form occur in June, July, August, September and October. This suggests the possibility of some other medium than the louse as an additional agent of transmission. . . . And the circumstance which would suggest that the body louse may have but little to do with the transmission of the endemic form of the disease, is the almost total absence of family infection. In five hundred cases which have been observed of this form of disease, the writer has seen only two instances in which another member of the family had been infected with endemic typhus at the same or nearly the same time. The transmissibility of the endemic form is practically nil. . . . Wherein does the second form of typhus which we have called the endemic differ from the epidemic form? They are alike in their onset, in the first stage of the eruption, in the critical decline, and neither is followed by relapse. In all other respects they differ.

Following on Brill's cases in New York and the cases first reported in Adelaide in 1921 there have been appearing in widely separated centres in the United States records of cases resembling mild typhus fever, in which a positive Weil-Felix reaction is

obtained. These infections are spread by another vector than the body louse.

In 1923 Kenneth Maxcy and Leon C. Havens[11] reported a series of eleven cases in Alabama in patients whose serum yielded a positive Weil-Felix reaction, similar to those described in Adelaide in their clinical manifestations. These occurred from June to December and in a footnote it is stated that more cases were being seen.

In 1925 Sinclair and Maxcy[12] reported a series of twenty cases of mild typhus in the lower Rio Grande Valley, occurring from July onward, with close association in certain cases, but no evidence of body louse infestation. There was the same failure with inoculation experiments. They say that while the "concept of indigenous cases of typhus fever was new to most of the American physicians, these mild cases of typhus had been recognized in Mexican towns (quite apart from "tabardillo") for ten years previously." They suggest spread by the head louse, though this is not proved.

In June Kenneth Maxcy[13] reports two hundred and nine cases of endemic typhus in the past three years in Alabama and Georgia with eight deaths. The description of these cases would answer for ours. He looks upon them as the same as Brill described in New York and adds:

> On the other hand, the epidemiology of the disease observed in southern United States presents certain differences from that of Old World typhus which suggest that the mode of transmission may not be the same; that there may be some mode other than direct transmission from man to man by means of the bite of a louse.

In December, 1926, Kenneth Maxcy[14] has another article on the same subject in which he mentions the successful transmission of the disease to *Rhesus* monkeys and guinea pigs. The maximum incidence is in the summer and fall (as with Brill and as with us). He states:

> These analyses of the occupations of persons attacked by endemic typhus suggest very strongly that as compared with the rest of the population those engaged in "trade" and especially those employed in food depôts, groceries, feed stores and restaurants, are exposed to a distinctly increased risk of infection.

And he sums up:

> The epidemiological characteristics afford no evidence suggesting louse transmission and are interpreted as being at variance with man-to-man transfer by lice, unless it be assumed at the same time that the disease occurs mostly in unrecognized form. . . . It is suggested as an hypothesis which seems to afford a more probable explanation of the mode of transmission that a reservoir exists other than in man and that this reservoir is in rodents, probably rats or mice, from which the disease is occasionally transmitted to man.

Thus all lines of evidence converge to demonstrate that all these groups belong to the same family and are in fact the same disease appearing in different quarters of the globe. Under these circumstances it seems justifiable to speak frankly of all our cases in Australia as endemic typhus fever and to carry on any further investigations on this basis.

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Reports of Cases.

AN UNUSUAL FOREIGN BODY.

By H. C. RUTHERFORD DARLING, M.D., M.S. (Lond.), F.R.C.S. (Eng.),
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ONE of our distinguished Sydney surgeons delights to tell the story of the top hat which an operator in a fit of mental aberration left in his patient's abdomen. I relate here the tale of a glove which after an operation found a resting place upon the patient's lung.

A.B., aged twenty-five years, a wasted youth of erethinic constitution, was admitted into the Coast Hospital on April 8, 1926, complaining of cough and progressive weakness following on an operation which he had undergone at another hospital one year previously.

There had never been any hæmoptysis. He knew of no family predisposition to pulmonary tuberculosis. The pulse was 120, the tongue furred, the temperature 38·2° C. (100·8° F.) and the respirations 28 per minute. The thorax was small, the muscles wasted and in the fifth right intercostal space just below the nipple was a curved three-inch surgical incision in the centre of which was a sinus about the calibre of a No. 6 English catheter emitting a free purulent discharge.

The expansion of the right side of the chest was deficient, the percussion note muffled and the breath sounds were masked over extended areas by diffuse, moist crepitant râles.

The left apex yielded a dull note to percussion, with definite cavernous breathing, but no adventitious sounds

could be heard. The note of the left base was impaired, but nothing beyond some harshness and feebleness of breathing could be detected. The sputum was mucopurulent, greenish in colour and on examination tubercle bacilli were found irregularly distributed through it.

Radiological examination demonstrated tuberculous mottling not only through the whole of the right lung field, but also to a lesser degree in the upper half of the left field.

The morning and evening temperature taken on four consecutive days showed a maximum morning height of 38·9° C. (102° F.), average 38·2° C. (100·8° F.) and maximum evening temperature of 39·3° C. (102·8° F.), average 38·3° C. (101° F.).

Exploration of the sinus by means of a probe failed to demonstrate the presence of any diseased bone. The probe could be inserted in a postero-superior direction for approximately 7·5 centimetres (three inches) when its further progress became arrested. During exploration of the sinus under local anæsthesia a piece of indiarubber was observed in the depth of the sinus. On further dilatation a cavity was located lying between the chest wall and the anterior surfaces of the upper and middle pulmonary lobes, containing a number seven and a half surgical glove and about 150 cubic centimetres (five ounces) of very offensive purulent fluid. The glove was extracted and a flanged rubber tube inserted down the sinus for the purpose of drainage.

The patient's condition progressively improved until April 19 when the limbs and trunk became covered with a profuse erythematous rash and the upper lip was greatly swollen. Thereafter the patient rapidly went down and finally succumbed on May 5, 1926.

PERNICIOUS ANÆMIA WITH SPLENOMEGALY.

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A WOMAN, aged fifty-two years, was admitted to Saint Vincent's Hospital on March 1, 1927. She gave a history of having had rheumatic fever at twenty-six years of age. She has had ten children and nine are alive and all are healthy. The family history contains nothing of any interest, except that one sister died after an attack of rheumatic fever. She has four other sisters and five brothers. All are alive and healthy.

The present illness began seven and a half years ago when the patient noticed numbness of the arm. Later she noticed that her skin was becoming yellowish and after a time she became breathless on slight exertion. She was in the Royal Prince Alfred Hospital six years ago for ten weeks and though improved in her general health, her condition was much the same on discharge. She continued to carry out the treatment in her home for five years. During the past two years the patient has been suffering from attacks of pain in her left hypochondrium, each lasting about a week. The pain is stabbing in nature, especially on movement. These attacks used to come on about every five or six months. She has had several periods of remission during which she would feel well. The longest of these was about three months and she would again relapse into her former condition. No attacks of vomiting or diarrhœa have occurred.

The patient lies comfortably in bed. A lemon-yellow tint is present on the skin and conjunctivæ. The teeth are all false. She had pyorrhœa and had all her teeth extracted some years ago. The tongue is slightly furred, it is neither sore nor tender, but somewhat glazed. The left side of the abdomen is more prominent than the right. The spleen is very enlarged and firm and extends one to two fingers' breadth below the umbilicus. The liver is slightly enlarged. Nothing abnormal is found in the respiratory system. The cardiac apex beat is not visible, but palpable and in normal position. The apex beat is snapping in character. Percussion reveals no increase in size. On auscultation a systolic murmur is heard over all areas, loudest over the apex and conducted well into the axilla. The first sound is heard at the apex and is slapping in character. The pulse is feeble, the rate is 88 per minute. The blood pressure is low; the systolic pressure is 120 and the diastolic 70 millimetres of mercury. The nervous system is clear. The plantar reflexes are normal. The knee jerks are present and normal. There is no evidence of any cord symptoms. No diminished sensibility or weakness is present in the limbs. The skin has a waxy lemon-yellow tint with a definite icterus. There is no pigmentation and no petechiæ are present. There is no œdema. No enlarged lymphatic glands are demonstrable.

The patient has some frequency of micturition especially at night. The urine contains a slight trace of albumin. Its specific gravity is 1020. It contains a few hyaline and epithelial casts. No pus and no red blood cells are present. It is sterile on culture. The range of urea concentration has been estimated by Calvert's test. The maximum urea is 2·05% and the minimum urea is 0·55%. This shows that kidney efficiency is very good. The blood urea is thirty milligrammes per hundred cubic centimetres.

The menopause took place some years ago. No vaginal discharge of any kind is present.

A fractional test meal revealed a very pronounced achlorhydria and almost a complete absence of free hydrochloric acid. No lactic acid and no Oppler-Boas bacilli were present.

On March 3, 1927, the erythrocytes numbered 1,500,000 per cubic millimetre. The hæmoglobin value was 45%, the colour index was 1·5. Numerous macrocytes, polychromasia, anisocytosis and pronounced poikolocytosis were present. Megaloblasts and nucleated red cells were found. The leucocytes numbered 10,000 per cubic millimetre. The polymorphonuclear cells were 60%, the lymphocytes 37%, eosinophile cells 1% and large mononuclear cells 2%. No myelocytes were present and nothing abnormal in character of lymphocytes was found.

On March 16, 1927, the Van den Bergh test gave an indirect (delayed) reaction.

On March 30, 1927, the blood count (after treatment with arsenic and dilute hydrochloric acid) resulted as follows:

Red cells, 2,050,000 per cubic millimetre;
Hæmoglobin value, 5%;
Colour index, 1·2.

Polychromasia was present. Macrocytes in fairly large numbers, megaloblasts and nucleated red cells were seen. The white cells numbered 5,800 per cubic millimetre.

On May 1, 1927, blood transfusion was carried out, five hundred cubic centimetres being given and two applications of X rays were made over the spleen at intervals of two weeks.

On May 4, 1927, the blood count revealed the following:

Red cells, 2,400,000 per cubic millimetre;
Hæmoglobin value, 60%;
Colour index, 1·2.

Polychromasia was present. Megaloblasts were present, but were few in number and some nucleated red cells were seen. The white cells numbered 9,000 per cubic millimetre. No myelocytes were seen and no characteristic change in nature of cells was found.

A few days after the blood transfusion and about a week after the second application of X rays to the spleen, the patient complained of pain in the abdomen on the left side. Her temperature went up to 38·3° C. (101° F.) for a few days and the spleen was very tender. Evidently this was due to an attack of some inflammatory disturbance in or around the spleen. The latter has diminished in size since the application of the X rays and is now only just below the costal margin.

When last seen the patient felt well and looked very much improved in appearance. The only treatment she has had apart from suitable diet and rest in bed for over eight weeks has been the administration of arsenic and dilute hydrochloric acid by mouth and the application of X rays over her spleen with one blood transfusion. I was anxious to try the effect of repeated blood transfusions. Professor Carmalt Jones, of Dunedin, has reported rather

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on May 12, 1927.

encouraging results with blood transfusion if the latter is frequently repeated. Unfortunately this patient belongs to one of the rare groups; we had to test six people before we could get one compatible and consequently in a hospital where it is difficult to arrange to get donors at any time, frequently repeated transfusions were hard to arrange, in fact we could not do so.

Comment.

The interesting feature of this case is the unusual size of the spleen which when the patient first came into the hospital before having irradiation, was more the size that is expected in a case of Banti's disease or leuchæmia. Although this is I think a definite case of pernicious anæmia in spite of the absence of the typical tongue, the absence of a constant leucopenia and any typical symptoms, it is certainly most unusual to meet with a spleen of this size. At the same time patients suffering from true pernicious anæmia are seen with a spleen below the costal margin. Such conditions generally have lasted for a considerable time as this one has. The main difficulty in diagnosis was the occurrence of such a large spleen and the variable total leucocyte count being above normal at times both of which may however occur in true pernicious anæmia. The alternative in diagnosis was that the condition might be an example of so-called Leube's leuchæmia, that is a condition of myelocythæmia to which a typical or more or less typical pernicious anæmia blood picture is added. We all know that sometimes in stages of remission of myelocythæmia and at times in the course of a leucanæmia, although the white cells usually give evidence of some of the characteristic cells even when their number is normal or just above normal or even below normal, the number of white cells may fall considerably either to just above normal or even below it. In other words there may be an actual leucopenia with no myelocytes *et cetera* at all and the blood film at the time may be exactly like that of pernicious anæmia, although *post mortem* appearances in these circumstances have been those of myelocythæmia. There is nothing, however, in this patient to justify the opinion that she is suffering from leuchæmia pure and simple. The white cell count has never been suggestive of this at all. Moreover the disease has existed a very long time for a myelocythæmia, especially too as she has improved greatly with treatment during the past four months. Again she has never had any glandular enlargement anywhere which, although uncertain, often occurs. Nor has she had any subjective symptoms associated with this condition. Finally, repeated blood examinations have always revealed a fairly typical pernicious anæmia film with not only megaloblasts, but a good deal of anisocytosis and a very large number of macrocytes. I must admit of course that many cases of aleuchæmia (pseudoleuchæmia and myeloid leuchæmia) run a very aberrant course and at times the only evidence as gauged by the films may possibly be that of a true pernicious anæmia. In such cases, however, the diagnosis can be made only by having repeated blood examinations done. Until evidence of even a suggestive characteristic blood picture is obtained, one is not justified, I think, in making a diagnosis of anything but pernicious anæmia, especially if the signs are fairly typical and consistent as they have been in this case, even though some unusual features are present. The blood picture of course definitely excludes Banti's disease (splenic anæmia).

SCARLET FEVER.

By G. B. DOWNES, M.B., Ch.M. (Sydney),
Resident Medical Officer, Royal Alexandra Hospital for Children, Sydney.

ON February 25, 1927, R.S., aged two years, was admitted under Dr. R. A. Green to the Royal Alexandra Hospital, as suffering from urgent laryngeal diphtheria. Having been feverish and cross for twenty-four hours, his breathing had become difficult three hours before admission and had grown progressively worse. The voice, however, was not greatly affected. On admission there was intense inspiratory stridor with recession of ribs and cyanosis. The uvula was enlarged and of dark blue colour, being partially covered with a membranous exudate. There was œdema of the soft palate, pillars of fauces and larynx and the epiglottis was tremendously enlarged. After two unsuccessful attempts at intubation tracheotomy was performed with immediate relief.

At this stage the diagnosis was regarded with doubt on account of the atypical appearance of the membrane, its absence from the trachea, the difficulty of intubation and the presence of the voice. The doubt was justified when a typical scarlet fever rash appeared the next day, followed by desquamation a week later. The tracheotomy tube was removed after forty-eight hours. Several throat swabbings failed to reveal any Klebs Löffler bacilli and the child made an uninterrupted recovery.

Osler states that the œdema is "usually slight or transient" but that it "may prove rapidly fatal." There have been at least two previous similar patients in this hospital successfully treated by tracheotomy.

Reviews.

ULTRA-VIOLET THERAPY.

MEDICAL practitioners contemplating the treatment of children with rickets, anæmia, malnutrition *et cetera* by artificial light will do well to digest thoroughly Dr. Katherine M. L. Gamgee's book on the subject.[1]

The author has produced an eminently useful and thoroughly practical account of the various aspects of the working of a clinic in any industrial area. Although fortunately at present Australians are not faced with the same bad conditions of housing and so forth that exist in many other parts, a clinic for treatment with artificial light would probably serve a very useful purpose.

The book is divided into four parts convenient for reference and making for easy reading. The printing is good and illustrations, radiograms, charts *et cetera* are profuse and excellently produced and a bibliography of references is given at the end of each chapter.

In the introduction Professor Leonard Hill points out that although good hygienic conditions are of first importance in preventing infantile disorders, still artificial sunlight has been of the greatest help in treatment. Also in the absence of ideal conditions ultra-violet radiation, particularly when combined with cod liver oil, has proved a specific for infantile rickets.

The author in her preface states that the object of her book is to set forth the salient features of artificial light therapy as applied to children in whom reactions present important differences to those of adults.

In the opening chapters the physics of the subject is briefly dealt with and more detail is given in a well arranged appendix. There is also a rough classification of the pathological states with which a clinic, such as that described by the author would be concerned.

One chapter is given to a description of rickets. The history, ætiology, diagnosis, pathology and treatment are dealt with briefly yet in a full and lucid manner, the work of modern research workers giving an almost fantastic touch when the effects of ultra-violet radiation on rickets are discussed.

Part II deals with the practical and administrative organization of a clinic and is full of practical advice as to the premises, equipment, staffing, routine and records, coordination and after-care.

In Part III the author discusses clinical and practical points such as estimation of height, weight and hæmoglobin percentage, dosage in children, the use of X rays in diagnosis *et cetera*.

Part IV consists of an analysis of case reports with a collection of histories.

Altogether this is a most compact and readable addition to the literature of ultra-violet therapy.

[1] "The Artificial Light Treatment of Children in Rickets, Anæmia and Malnutrition," by Katherine M. L. Gamgee, M.R.C.S. (England), L.R.C.P. (London), D.P.H. (R.C.P.S.), with an Introduction by Professor Leonard Hill, M.B., F.R.S.; 1927. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 192, with illustrations. Price: 10s. 6d. net.

The Medical Journal of Australia

SATURDAY, AUGUST 13, 1927.

A Problem in Medical Education.

THE three medical schools attached to the Universities of Sydney, Melbourne and Adelaide provide excellent teaching for medical students. It is true that the curricula are based on a haphazard principle and that they should be relinquished in favour of a logical and efficient curriculum built up with due regard to the requirements of modern scientific medicine. The medical schools and bodies comprised of acknowledged leaders of medical thought have instituted post-graduate courses in some special branches of medicine both for general practitioners and for those who wish to specialize in tropical hygiene and public health work. The Universities grant higher degrees in medicine and in surgery, but there is little teaching of an advanced kind. The degrees of doctor of medicine and of master of surgery are granted after the submission of theses and an exhaustive examination or other test of extensive general knowledge and high culture is not carried out. The degree of doctor of medicine may be gained as the result of a thesis on work of a purely surgical nature. The candidate at times exhibits no evidence that he possesses knowledge distinguishing him as a learned physician. In these circumstances many young graduates with ability, ambition and some means travel to England and seek to obtain the membership of the Royal College of Physicians of London. While it may be admitted that the mere possession of a degree or diploma does not necessarily prove that the holder is an exceptionally learned or gifted person, it is at present the only indication, other than that indefinite quality known as reputation, of special study and possession of knowledge. It is safe to assume that a practitioner with a diploma of the membership of the Royal College of Physicians of London is a competent, diligent and learned physician and that he who has been elected a fellow of the College, possesses these qualities to an exceptional degree.

There is no institution analogous to the Royal College of Physicians of London in Australia. The College of Surgeons of Australasia has recently been founded, but this body is still too young to undertake the responsible task of standardizing the teaching and prescribing the minimum requirements for a higher degree in surgery. If anything is done to remedy the defect, it must emanate from the medical schools. The three medical schools have been in being for a sufficiently long time to claim recognition as first-class teaching centres. They have justified their existence as training schools for medical students. That the teachers in the schools are competent to carry out advanced work will not be questioned. There is, therefore, no reason why our own medical schools should not undertake the tasks that are fulfilled at present in London.

In making this demand, we would distinguish between the ordinary post-graduate courses for general practitioners which are held annually, and the courses of advanced teaching. The main objective of the advanced courses should be educational; the passing of an examination should remain a matter of secondary importance. The classes should be continuous throughout the academic year and should be so organized that those participating could be certain of the opportunity of covering the whole field of medicine. If the teaching were well planned and proper facilities were created, the graduate of a few years' standing could add to his store of knowledge and experience to enable him to qualify for the post of teacher, for the rank of consultant and for the office of physician at the great metropolitan hospitals. In order to extend this experience he could undergo the advanced course of study at a school other than the one in which he spent his student days. It might be advisable to mould the course in such a manner that while some graduates would devote the whole of their time for a year or two to this study, others could attend some of the classes while carrying out remunerative work. All the arrangements should be made with the view to giving the best service to Australian graduates. It should be

unnecessary for anyone to go to England to complete his medical education. Post-graduate work in Great Britain and on the continent of Europe will always remain a very valuable asset, but it should not be a necessity. It would be essential for a full measure of success for the authorities of the three medical schools to adopt similar lines of action and to introduce a system of reciprocity and interchange of advanced graduate students. The fees for attendance should be low, for one of the objects would be to provide facilities for the extension of the knowledge of those who cannot afford to spend a year or more on the other side of the world. We commend the scheme to the serious consideration of the Faculties of Medicine of Sydney, Melbourne and Adelaide.

Current Comment.

ACHYLIA GASTRICA AND HISTAMINE.

THE subject of *achylia gastrica* has been discussed in these columns on more than one occasion. In August, 1926, attention was drawn to observations by Knud Faber on the pathogenesis of the condition. Faber holds that achylia has an exogenous cause, that external factors act on the stomach either by direct irritation of the mucous membrane or through the circulation by a toxic action on the gastric parenchyma. He believes that in this way the histological changes characteristic of gastritis are brought about and that eventually atrophy of the gastric glands occurs. It is well known that achylia may be apparent in the presence of a gastric mucosa whose glands present practically a normal appearance. Einhorn reported recovery from chronic achylia. It is obvious that recovery cannot occur if the glands are atrophied and it is not possible to subject to microscopical examination the gastric mucosa of a patient whose condition is being investigated. It thus becomes necessary to have at hand some means of determining whether the action of the gastric glands has been permanently suspended or, if Faber's views are accepted, whether the damage to the gastric glands has advanced to such a stage that recovery is impossible. In our issue of February 5, 1927, the significance of gastric anacidity was discussed in these pages in the light of work by Keefer and Bloomfield. These workers emphasized the value of the fractional test meal, but they also made some observations with the aid of histamine injected subcutaneously. They injected this substance into eight patients who were regarded as suffering from "functional" anacidity and six of them were found to yield free acid.

Histamine or iminazolyl ethylamine is derived from the amino acid histidine by a process of decarboxylation. Hanke and Koessler have shown that the formation of histamine is always coincident with the production of a medium that is distinctly acid. They believe that histamine is formed by the *Bacillus coli communis* to neutralize excess of acidity. It has been shown that histamine is a normal constituent of the caecum and colon in amounts sufficient to produce toxic symptoms if it were absorbed into the circulation. Hanke and Koessler hold that histamine is rendered pharmacologically inert in its passage through the intestinal wall. When it is injected subcutaneously, it causes a fall in blood pressure, dilatation of the superficial blood vessels and an increase in gastric secretion. In larger amounts it causes shock.

H. L. Bockus and Joseph Bank have carried investigations with histamine to a point further than that reached by Keefer and Bloomfield.[1] They have studied its effect when it is given simultaneously with a meal of bread and water and have compared the findings thus obtained with the gastric response as estimated by the fractional test meal. In all their experiments one milligramme of histamine (one cubic centimetre of a one in a thousand solution) was used for subcutaneous injection. They refer to the work of others on histamine in the diagnosis of *achylia gastrica*, but hold that no other observers have studied the effect of histamine on the stomach during the digestive phase with a carbohydrate meal. Twenty-one patients with "so-called achlorhydria" were examined. The diagnoses had been made after fractional gastric analysis on account of the absence of mineral acid throughout the two hour phase of gastric digestion. The patients after examination were placed into one of three groups. The first group comprised those with true *achylia gastrica*. In these no hydrochloric acid or enzyme was discovered by fractional or histamine analysis and no excretion of neutral red occurred into the stomach after its intravenous injection. The second group comprised those with achlorhydria. In these no mineral acid was found after fractional or histamine analysis, but a trace of enzyme was found following the fractional or histamine analysis or both. The third group comprised those with hypochlorhydria. In these subnormal amounts of mineral acid and enzyme were found after injection of histamine. Of the twenty-one patients eleven were found to belong to Group I, one was found to belong to Group II and nine to belong to Group III. The findings on injection of neutral red confirmed those obtained by histamine in every case in which it was used with one exception. In this instance the injection of neutral red was made into the muscular tissue. A comparison was made of the findings from fractional analysis and histamine injection in twenty-nine patients held to be suffering from hypochlorhydria. Histamine caused a definite increase in the acidity throughout the period of gastric digestion. It caused mineral acid to appear earlier and it prolonged the period of secretion. In eleven patients with normal acidity the injection of histamine caused an elevation of the degree of acidity, but

[1] *Archives of Internal Medicine*, April 15, 1927.

to a lesser extent than in patients with reduced acidity. When histamine was injected into eight patients with hyperacidity, the stomach did not respond to the drug. No definite pathological condition was diagnosed in any of the patients of the last mentioned group and none of them suffered from gastric stasis. The findings are regarded as supporting Carlson's contention that the stomach cannot secrete an acid of more than normal concentration.

The first conclusion of Bockus and Bank is sweeping, but must be accepted in large measure as true. They state that the great bulk of literature on *achylia gastrica* is worthless and that it is likely that only a small proportion of the cases reported as being of that nature during the last few years, were really cases of *achylia gastrica*. Their second conclusion is an obvious corollary, namely, that the diagnosis of *achylia gastrica* is impossible when the single extraction method is used. This has long been recognized and, as already mentioned, was emphasized by Keefer and Bloomfield. Bockus and Bank also state that in the present state of knowledge a diagnosis of *achylia gastrica* should not be made until it has been confirmed by the histamine test. They do not think that the histamine test should displace fractional gastric analysis. They hold with Rehfuss that a definite load must be imposed on the stomach in order that its ability to perform its function may be judged. The normal physiological load is food. Fractional gastric analysis and the histamine test will yield more information than either alone in stomachs with low acidity.

While there is no doubt that this work will be useful from the everyday point of view, it is a pity that Bockus and Bank have not discussed more fully the mechanism of the action of histamine on the gastric mucosa. They record the views of others, but do not give their own. Rothlin and Grundlach maintain that the gastric action of histamine is the result of vagus stimulation. Koskowski and Popielski believe that it acts directly on the gastric cells. Gutkowski found a gastric response after the subcutaneous injection of the drug into the posterior extremity of an animal whose only communication with the rest of the body was vascular. Ivy and Janois held that the mechanism is not nervous, because they succeeded in producing histamine secretion in a denervated Heidenhain pouch. Textbook authors state that the mechanism is not clear. One or two facts, however, can be remembered. In the first place the changes produced by histamine resemble those following shock. It is believed that in shock some of the blood plasma is extravasated, leaving the blood vessels rich in corpuscles. This extravasation actually occurs in histamine shock and may be demonstrated either by measurement of the concentration of hæmoglobin or by estimation of the corpuscles. In the second place the action of histamine on the gastric mucosa resembles the hormonic action occurring in normal digestion. The mechanism by which the hormones or "chemical messengers" produce their effects is not known, probably either vascular or nervous channels or both are used. The vascular changes following injection of histamine are most likely of vasomotor origin. The physical and chemical constitution of the blood supplying the gastric cells and of the intercellular fluid surrounding them would thus be altered and a corresponding intracellular change would take place. This intracellular change would also be in part the result of what might be called an hormonic call from other points in the alimentary canal. In this way the findings of Bockus and Banks could be explained. If the damage to the gastric cells had produced atrophy, they would not respond at all. Slightly damaged cells would respond to a certain extent and normal cells would become more active.

TUBERCULOSIS OF THE TONGUE.

In the course of an article published in 1924 Morrow and Miller reported that of 1,444 persons affected by tuberculosis a little less than 1% gave evidence of tuberculous involvement of the tongue. The latter condition is therefore to be regarded as comparatively rare. In 1916 J. R. Scott reviewed the literature of the subject and collected 231 cases. William H. Feldman has collected a further series of fifty-two cases and has reported in full the history of a patient who came under his care.[1] Tuberculosis of the tongue may be primary or secondary. Twenty-six of Scott's infections and thirteen of those collected by Feldman were regarded as primary. The latter uses the term primary in the "restricted" sense, meaning that it was impossible from the available data to discover a primary lesion in any other part of the body. It is obvious that the question is difficult of determination and the primary incidence is in all probability much less than the figures would indicate. The tongue of patients suffering from pulmonary tuberculosis like the lower part of the gastro-intestinal tract, is exposed to fluid containing tubercle bacilli. It is probable that infection takes place as a result of a breach of continuity of the mucous membrane. In the patient reported by Feldman involvement of the tongue followed the biting of the tongue; the patient suffered from pulmonary tuberculosis. Feldman admits that the lesion is usually the result of trauma, but he raises the question as to why males are affected so much more frequently than females. Of the fifty-two patients discussed by him forty-three were males. Most authors hold that the tongue of men is more exposed to trauma than that of females. Dental caries and sharp jagged teeth, injury from tobacco chewing and smoking have been put forward as factors influencing the high incidence in males. Feldman holds that there is not sufficient proof to justify this view. He is tempted to postulate a sex susceptibility, but is content to allow this to remain a matter of conjecture. The same question has been raised in connexion with malignant disease of the tongue. In the present state of knowledge it is wiser to conclude that the tongue of the male is more exposed to trauma and other influences leading to a lowered resistance than that of the female.

[1] *The American Journal of Pathology*, May, 1927.

Abstracts from Current Medical Literature.

RADIOLOGY.

Iodized Oil in Gynæcology.

Q. U. NEWELL (*The Radiological Review*, January, 1927) uses "Iodipin" (40% iodine in vegetable oil) for the exploration of the uterine cavity and tubes. A Grave's bivalve speculum is used and the field is painted with iodine. A sound is introduced into the uterus and when no obstruction is noted, it is withdrawn and a rubber tube with a collar is passed for two centimetres into the cervical canal. About seven cubic centimetres of "Iodipin" are slowly injected and the X ray pictures are made. In sterility when the tubes are found obstructed, it is possible to demonstrate the obstruction. When masses are present in the pelvis, the uterus can be distinguished from these masses. The size of the uterus and tubes may also be demonstrated, while any encroachment in the uterine cavity by fibromata *et cetera* may be demonstrated.

Differential Diagnosis of Bone Tumours.

H. W. MEYERDING (*Journal of the American Medical Association*, February 5, 1927) discusses the preoperative diagnosis of bone tumours. He classifies the various bone diseases so that the tumours progress from the inflammatory type through the benign to the malignant types as follows: (i) inflammatory, (ii) *osteitis fibrosa cystica* and cysts, (iii) benign tumours, (iv) giant-celled tumours, (v) angioma, (vi) endothelioma, (vii) periosseous fibrosarcoma, (viii) osteogenic sarcoma, (ix) multiple myeloma, (x) metastatic tumours. A carefully taken history and X ray examination will differentiate between most types, but some cannot be diagnosed until sections are examined or metastasis has occurred. Ossifying hæmatoma is suspected after injury with localized swelling, the new bone being laid down parallel with the shaft. Brodie's abscess involves the diaphysis and produces considerable local enlargement. A cystic area is seen in the skiagram and its margins may be sclerosed with some periosteal thickening. Giant-celled tumours may give a similar appearance, but usually the margins are less defined and the epiphyseal line invaded. Central sarcoma progresses rapidly with constant pain. Tuberculous lesions are seldom associated with severe intermittent pain, while muscle spasm, atrophy, joint involvement and stiffness are present. Callus formation tends to disappear, while new growth increases. *Osteitis fibrosa cystica* is slowly progressive with rheumatic-like pain; it occurs in young subjects and does not involve the epiphysis. Exostosis occurs in the young and consists of bone, cartilage and fibrous tissue. There is no pain and slow enlargement over years is common. Chondroma occurs in the medulla, is of slow growth and localized. Giant-celled tumours occur in youth and old age and usually near a joint. Fracture from slight trauma is frequent. Endothelioma resembles osteomyelitis, but the bone is expanded and contains rarefied areas. Epiphyses may be involved and metastases occur. Periosseous fibrosarcoma is accompanied by pain, is palpable and localized. It causes bone absorption by pressure, but no new bone formation. Osteogenic sarcoma occurs in adults and the pain is severe and constant with a short history; anæmia and loss of weight and strength occur early. Local heat and venous engorgement occur. Multiple myeloma presents little difficulty from the numerous tumours of the ribs, sternum and skull which occur.

Foreign Bodies in the Œsophagus.

W. F. MANGES (*American Journal of Roentgenology*, January, 1927) considers the diagnosis of foreign bodies in the œsophagus. The most common site for lodgement is at the level of the suprasternal notch. Coin-shaped foreign bodies are most commonly seen and they occur more often in children, while in adults the smaller types of foreign body are found which become embedded in the mucous membrane. Both fluoroscopic and radiographic examination are necessary and should include the whole alimentary tract. All clothing should be removed and only the best quality films accepted. In the detection of non-opaque foreign bodies, the screen examination is of greatest importance. Normal findings should not be recorded from X ray examination alone. The examination should be made as close to operation as possible and the patient should be examined after the removal of the foreign body. The greatest width of the œsophagus is in the lateral direction, while that of the trachea is in the antero-posterior direction. For large non-opaque foreign bodies in the œsophagus a thick watery solution of bismuth is given and this is held up by the foreign body. In some cases a small wet bismuth capsule is given. A dry capsule is likely to adhere to the wall of the œsophagus. Care must be exercised with foreign bodies of glass and capsules of bismuth and cotton wool should not be used.

Duodenal Ulcer.

C. G. SUTHERLAND considers the subject of duodenal ulcer (*Radiology*, February, 1927). In the Mayo Clinic duodenal ulcer is the most common of all lesions of the alimentary tract which are submitted to radiological examination. Duodenal ulcer, cholecystic disease and appendicitis are just as difficult to differentiate from one another radiologically as they are clinically. The pathognomonic radiological sign of duodenal ulcer is the *incisura* which deforms the normal contour of the bulb; this may be unilateral or bilateral and may vary in size from a mere slit on the lesser (or greater) curvature to an indentation of both contours that nearly bisects the cap. Of all duodenal ulcers 95% occur in the first part and usually they are close to the pylorus. Duodenal pouches near Vater's papilla are not uncommon and they also occur in the duodenal ampulla. Diverticula and ulcer niches persist, while usually the pouches can be emptied by manipulation. Periduodenal adhesions cause various bulb deformities. When gastric hypersecretion and dilatation are present, it is a good plan to let the patient lie prone for five minutes and then the meal will be seen to pass through a deformed duodenal bulb. Retention with hyperperistalsis and no gastric or pyloric defect is a definite sign of duodenal ulcer. The author calls attention to two signs found at operation which definitely point to the presence of ulcer: (i) scar with or without exudate and (ii) stippling of the serosa

Myelomatosis.

E. S. BLAINE (*The Radiological Review*, January, 1927) writes on myelomatosis and reports three cases. This condition is also known as *mollities ossium*, senile osteomalacia, myelogenous pseudoleuchæmia and multiple myeloma. It is apparently a specific infection of the bone marrow which invades the cortical bone and the lesions are invariably multiple. Albuminuria is almost constant. Pathologists incline to the view that the lesion is a low grade malignant growth. It occurs between thirty-five and fifty-five years of age and is slow in progress. Fractures from slight violence are common. The ribs and sternum are very common sites. Later extensive involvement of the bone marrow occurs with cachexia. Pain occurs when the periosteum is involved and the Bence Jones albumosuria occurs. The X ray examination is usually undertaken for a spontaneous fracture and there is a striking appearance of the abnormal bone structure in the immediate neighbourhood. There are numerous areas of sharply circumscribed loss of density, generally with rounded edges. In most instances the periosteum is intact. In the diffuse type an apparent osteoporosis spreads generally over a flat bone, notably the skull.

Subdiaphragmatic Abscess.

F. W. O'BRIEN discusses the X ray diagnosis of subdiaphragmatic abscess (*Boston Medical and Surgical Journal*, March 31, 1927). Early diagnosis is of great importance. Two types are seen, namely the so-called simple type and the gas-containing abscess. The abscess is unilateral and its location depends upon the source of infection. Hepatic, appendiceal and duodenal lesions usually lead to right-sided abscesses, while gastric, pancreatic and splenic lesions form abscesses on the left side. Abscess may occur at any age. X ray examination reveals elevation of the diaphragm on the affected side and fixation, in gas-containing abscess the level of fluid may be noted in the abscess with the gas area above it.

PHYSICAL THERAPY.

Ultra-Violet Light in the Treatment of Ulcers of the Leg.

A. E. SCHILLER (*Archives of Physical Therapy, X-Ray and Radium*, February, 1927) divides ulcers of the varicose type in the leg into those due to the superficial varix, in which case the ulcer lies in close relation to the varicose vein, those due to the surface varix complicated by the varicosity of the perforating veins which tend to appear near the perforating veins, and postphlebitic ulcers which always appear within two years and usually within six months after the phlebitis and are recognized by the presence of small, hard, straight veins in the thigh and calf. Syphilis and trauma are the most important ætiological factors and the streptococcus is the principal pathogenic agent. It is necessary to differentiate between ulcer of the varix type, simple ecthyma, ulcerative syphilides and tuberculous ulcers. Patients with hyperglycæmia not accompanied by glycosuria rapidly recover under treatment with "Insulin," ten units being injected subcutaneously twice a day. The first requisite is to clean the surface of the ulcer and remove the dead epithelium. Ultra-violet light of short wave length focally applied kills the superficial organisms and if used in conjunction with mercurochrome, the deeper ones as well. It has definite sedative action and stimulates resorption. Regional application of light of longer wave lengths stimulates the circulation and increases local metabolism. It meets the pathological requirements of these ulcers more satisfactorily than any other treatment. Cure of the ulcer must be followed by care of the leg. Either the veins must be removed or elastic stocking supports for the leg be worn constantly to prevent recurrence.

The Effect of Diathermy on Gastric Acidity.

LOUIS J. BRADY (*Archives of Physical Therapy, X-Ray and Radium*, March, 1927) reports an attempt to produce *achylia gastrica* in dogs by means of diathermy. The dog was anæsthetized with ether and given 0·01 gramme of morphine subcutaneously. A large electrode was attached to a shaven area ten centimetres wide around the abdomen just below the diaphragm and a small rounded electrode was passed into the stomach. It was found that medium voltage up to a thousand milliampères could be applied for as long as sixty minutes with safety. If a current of five thousand milliampères of low voltage was applied, œdema and burns could be seen in the gastric mucosa. The results were very surprising, as a considerable increase in the total and free acidity occurred immediately after treatment. This was soon followed by a fall in acidity to be followed by another increase after renewed treatment. The longer the periods of subjection to diathermy and the shorter the interval between the treatments, the shorter was the period of acid depression. By using a small initial treatment of two hundred milliampères and by increasing the amount each day by one hundred milliampères until a dose of five hundred milliampères was given, the acidity was found to rise steadily until a definite point was reached. This suggests a method of treating a vast number of patients with gastric achylia of neurogenic or idiopathic origin. By using an anterior and a posterior plate it was found possible to raise the gastric temperature to 41° C..

The Radiation Treatment of Genital Carcinoma.

OTTO VON FRANQUÉ (*Strahlentherapie*, 1926, *Band* 21) states that present-day statistics seem to favour the use of radiation therapy over surgery in the treatment of genital carcinoma. The unfavourable results are attributed to improper technique, especially overdosage. Experience shows that an average dose of 6,000 radium element milligramme hours is required to effect a cure. At the Radium Institute, Stockholm, the dose is 6,700 milligramme hours. At the Bonn clinic the dose is 6,000 milligramme hours and in America (the average of twenty-two hospitals) 5,000 milligramme hours. The results in operable tumours are as good as those of surgery. Care must be taken to avoid premature termination of the treatment. Improvement occurs within two or three months, but cessation of hæmorrhage and increase of weight should not be looked upon as signs of a cure. One hundred milligrammes of radium element are applied intracervically for thirty-six hours on two occasions and a complete Röntgen treatment is given through large fields in the abdomen, back, both sides and vulva. The whole treatment cannot be repeated until six months have elapsed and during that interval surgery should be adopted if necessary for the operable tumours. The Röntgen treatment may be repeated in three to six months. Radiation treatment has a mortality of 1%. Carcinoma of the body of the uterus is curable by radiation with more certainty than that of the cervix, if it has not advanced too far. Radium seeds are not so simple to use and increase the risk of the treatment.

Röntgen Treatment of Intraspinal Tumours.

R. LEDOUX LEBARD AND ÉTIENNE-PIOT (*La Presse Médicale*, April 13, 1927) hold that the intraspinal injection of "Lipiodol" for the exact localization of intraspinal tumours will be the means of avoiding useless surgery such as occurred in 18% of patients with intraspinal tumours at the Mayo Clinic. Although radiation offers a prognosis equally as favourable as surgery, it is an advantage to combine the methods. Laminectomy is followed by complete or partial extirpation and when the wound is healed, radiotherapy is begun. One-sixth to one-third of a dose is administered every two or three days until 4,000 X ray units (French) have been given. Two hundred kilovolts, one millimetre of copper as a filter, twenty-eight centimetres distance and a port or ports of entry twelve to twenty centimetres square are the factors employed. Four cases are reported in detail. A patient with a perithelial sarcoma treated by the combined method was quite well after three years. Another with lymphosarcoma, also treated by the combined method, was well after two and a half years. A third patient with an intramedullary tumour, probably a glioma, was treated by radiation only, and was clinically well after twelve months. The fourth patient with an angiomatous deformity of the cord manifested slight improvement only. These figures are held to be superior to any others published. The advantage is due to the combined method, high voltage and heavy infiltration.

Ultra-Violet Light Therapy in Whooping Cough.

BRU CAMILLE (*Journal de Radiologie et d'Électrologie*, April, 1927) employed ultra-violet light during an epidemic of whooping cough. He found among children of all ages and conditions that 80% were cured and 10% improved, while the remaining 10% manifested no effect. Treatment was carried out by two minute exposures to the back and front at seventy centimetres distance on the first day. Treatment was given every second day and an increase of two minutes was made at each exposure. Erythema occurred nearly always after the third treatment and was accompanied by a decrease in the number and violence of the attacks, lessening of the fatigue, diminution of vomiting and improvement of the general condition. The percentages of cures is a definite improvement on that associated with other remedies. The number of coughs is lessened and when they occur, they respond to ultra-violet light.

Small Intestine Radiography.

R. W. MORSE AND L. G. COLE (*Radiology*, February, 1927) describe a Röntgen method of examining the small intestine. An opaque meal, consisting of two hundred and forty grammes (eight ounces) by weight of barium sulphate mixed to a paste with one hundred and fifty cubic centimetres (five ounces) of water giving a bulk of two hundred and ten cubic centimetres (seven fluid ounces) is used. The stomach and duodenum are examined by the serial film method and then large films are used for studying the small intestine. Films are usually taken half an hour after the meal and at two, four and six hours. More frequent films are taken if necessary. In this way a record may be obtained of the contour of the inner surface, the state of the lumen and the position of the intestine. The small intestine is normally in a state of tonic contraction and its appearance depends on the degree of distension. Rapid uniform emptying of the stomach gives a large calibre to the small bowel throughout its length. Intermittent gastric evacuation will indicate irregular calibre of the intestine.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at Saint Vincent's Hospital, Melbourne, on June 15, 1927. The meeting took the form of a series of clinical demonstrations by the members of the honorary staff.

Hydatid of the Muscles of the Thigh and of the Liver.

MR. H. B. DEVINE showed a male patient, aged thirty-two years. Fourteen years previously he had been kicked on the thigh which had remained stiff for one week. About two years later the thigh had begun to swell and since then had been gradually increasing in size. Two weeks previously while chopping wood, he had noticed at the centre of the right costal margin, a sharp pain which had been made worse by yawning, but not by deep breathing. There had been a slight cough, but no sputum. He had been jaundiced for one week. The temperature had been slightly raised. On examination there was dulness and diminution of the breath sounds, vocal resonance and fremitus at the base of the right lung and in the right axilla. The lower margin of the liver was palpable. In the region of the right thigh there was a fluctuant swelling about the size of a football. A skiagram revealed extensive mottling surrounding the femur and extending for some distance into the mass. The outline of the bone appeared to be distinct. The Casoni test for hydatid disease had yielded a positive result. Mr. Devine thought that there was a hydatid cyst in the liver and also in the muscles of the thigh.

Gastro-Enterostomy.

Mr. Devine also showed a cinematograph film illustrating the operation of gastro-enterostomy.

Ovarian Auto-Transplantation.

DR. A. NORMAN MCARTHUR showed a female patient, aged twenty-six years, who had been complaining of premenstrual ovarian pain. Three months previously an autogenous ovarian transplantation had been performed. Since the operation she had not menstruated and still had pain over the site of the left ovary. From past experience Dr. McArthur still expected that menstruation would occur, and possibly pregnancy as in former cases recorded by him.

Uterine Hæmorrhage.

Dr. McArthur also showed three uteri which had been removed for excessive hæmorrhage. In two of these cases curettage had been performed elsewhere, but nothing had been removed by the curette and the hæmorrhage had still continued. Both uteri showed the flat form of hypertrophied endometrium of the type of the "shawl stalactite" with long tags which the curette could not engage.

The third specimen had been removed from a female, aged forty-eight years, who ten years previously had had a myomectomy performed. An examination of the uterus had, as yet, shown no cause why hæmorrhage should have occurred.

The Results of Various Operations for Fractures.

MR. C. GORDON SHAW showed a male patient, aged sixty-seven, on whom he had operated two years previously for an intracapsular fracture of the neck of the right femur which had been ununited for twelve months. A fibular peg had been used. At the time of presentation the function of the limb was almost perfect and there was only one centimetre of shortening.

Mr. Shaw also exhibited two patients in whom a spinal fracture of the tibia and a fracture of the radius had been treated by Lane's plating with a good result in each instance.

After Result of Excision of the Knee Joint.

DR. J. FORBES MACKENZIE showed a male patient, aged seventy years, on whom an excision of the right knee joint had been performed for a septic arthritis of two years' standing. A good functional result had been obtained.

Typhoidal Perichondritis.

Dr. Mackenzie's next patient was a male, aged forty, who had had an attack of typhoid fever in 1926, following which he had developed perichondritis in the region of the sixth right costal cartilage. The diseased cartilage had been excised and vaccine treatment instituted.

Ruptured Crucial Ligaments.

Dr. Mackenzie's last patient was a male, aged fifty-three years. About two and a half years previously he had had a dislocation of the left knee joint with rupture of the crucial ligaments. Following this the joint had gradually increased in size and at the time of presentation simulated very closely Charcot's disease. The joint contained "loose bodies" and presented the usual features seen in the absence of both crucial ligaments. The blood had failed to react to the Wassermann test and a careful examination had revealed no signs of syphilis. Dr. Mackenzie thought that the joint condition was due to chronic inflammation supervening on the injury.

Surgical Methods.

Dr. Mackenzie also demonstrated some surgical methods in the treatment of carbuncles, fractures of the clavicle and hæmorrhage from loose-walled cavities. He considered that, when incising a carbuncle, the incision should be carried through the deep fascia into the muscles beneath.

A method of peritoneal suture was also demonstrated.

Muscular Dystrophy.

DR. J. W. GRIEVE showed a female, aged twelve years, who was the fourth of five children, all the others being well. She had had asthma and chronic bronchitis for many years. Three years previously she had had measles and whooping cough. At the age of three and a half years her mother had noticed weakness and thinness of the muscles of the face and about twelve months ago weakness and wasting of the shoulder girdle muscles had been observed. At the time of presentation all the muscles of the face and shoulder girdle manifested marked weakness and wasting and diminished response to faradic stimulation.

Hæmochromatosis.

Dr. Grieve's next patient was a male, aged fifty-three years. For several years his skin had been bronzed. Three months previously he had noticed thirst and polyuria. He had been admitted to hospital on April 7, 1927, when his urine had contained sugar and diacetic acid and the blood sugar curve had been of the typical diabetic type. His weight had been fifty-three kilograms (eight stone four pounds). The liver had been palpable one hand's breadth below the costal margin. After his admission to hospital he had developed ascites and œdema of the legs which had since improved considerably. At the time of the meeting he was on a diet containing 45 grammes of carbohydrate, 67 grammes of protein and 135 grammes of fat and was receiving 20 units of "Insulin" three times a day. The urine contained no sugar or diacetic acid and his weight was sixty-five kilograms (ten stone three pounds).

Cretinism.

Dr. Grieve also showed a boy, aged fourteen years, who was the eldest of four children, the other three all being well. His mother's features were typical of slight myxœdema. His development had been regarded as being normal until he was seven years old, when it had been noticed that a brother two years younger was "catching up" to him. He had always been considered fat, but just before he had attended hospital in September, 1926, he had been getting stouter and slower mentally. His weight had been thirty-four kilograms (five stone five pounds), the average for his age being forty-one kilograms (six stone seven pounds), and his height one hundred and twenty-seven centimetres (four feet two inches), the average being one hundred and fifty-two centimetres (five

feet). The blood had failed to react to the Wassermann test. He had been given thyreoid extract six centigrammes (one grain) three times a day and the dose had been gradually increased to twelve centigrammes (two grains). In nine months he had increased nine centimetres (four and three-quarter inches) in height and had gained 4·3 kilograms (nine and a half pounds) in weight. He was much brighter mentally.

Encysted Pleural Effusion.

Dr. Grieve's last patient was a male, aged sixteen years. For the past seven months he had had attacks of sharp pain in the right axilla, occurring about once a week and lasting two days. There had been no cough, but on taking a deep breath he had noticed a "grating" feeling in the right side of the chest. The temperature had been slightly raised. In the right axillary region there had been dulness to percussion and slight swelling. The percussion note anteriorly had been good, but was rather impaired posteriorly. A needle had been inserted through the dull area, but no fluid had been obtained. It was thought that the condition was possibly an encysted pleuritic effusion.

Cerebro-Spinal Syphilis.

DR. THOMAS KING showed a female, aged thirty-five years. For the past three years she had had shooting pains in the legs. The pupils did not react to light, the knee jerks were absent and both the blood and the cerebro-spinal fluid had reacted strongly to the Wassermann test. Intravenous injections of "Novarsenobenzol" had been given, but no improvement had resulted. Lumbar puncture had been performed and twenty cubic centimetres of cerebro-spinal fluid which was under increased pressure, had been removed. An examination of the fluid had revealed a trace of globulin and sixty-five leucocytes per cubic millimetre, 95% of which were lymphocytes. Five cubic centimetres of a solution containing 0·15 gramme of "Novarsenobenzol" in three hundred cubic centimetres of water had been injected intrathecally. On the following night the patient had complained of severe pains in the legs, which had not recurred since except immediately following a second injection. Dr. King considered that the changes in the cerebro-spinal fluid suggested a good deal of vascular involvement of the spinal cord which accounted for the apparent cure of what was probably syphilitic meningomyelitis.

Dr. King also showed a male, aged thirty years, who was suffering from *tabes dorsalis*. The cerebro-spinal fluid manifested no evidence of interstitial changes, as in the first patient. Both the blood and the cerebro-spinal fluid had reacted strongly to the Wassermann test. Five cubic centimetres of a solution containing 0·15 gramme of "Novarsenobenzol" in fifty cubic centimetres of water had been injected intrathecally and twelve hours later the patient had complained of severe pain in the legs and subsequently developed a complete paralysis of the lower limbs and sphincters which at the time of the meeting was beginning to improve. Dr. King pointed out that this injection was six times as strong as that given to the first patient and apparently too concentrated.

Syphilitic Disease of Bone.

Dr. King's next patient was a male, aged twenty-nine years. A small, hard, tender tumour could be felt on the ventral aspect of the upper third of the right humerus. The blood had reacted strongly to the Wassermann test. A skiagram revealed gummatous destruction of bone at the site of the tumour. Injections of "Novarsenobenzol" and bismuth had been given and also iodide of mercury by the mouth, but no improvement had resulted.

Hydronephrosis and Double Ureters.

Dr. King also presented a female patient who had had typical attacks of renal colic accompanied by hæmaturia. X ray examination of the urinary tract had revealed no abnormality and a few days later a uric acid calculus about the size of the tip of the finger had been passed. Attacks of renal colic and hæmaturia had continued and a further X ray examination had revealed in the region of the renal pelvis a shadow which, it was thought, might have been due to a uric acid calculus. The ureter had been catheterized and a solution of sodium iodide injected. The capacity of the renal pelvis had been twenty-five cubic centimetres. The pyelogram revealed some dilatation of the renal pelvis and a double ureter. Dr. King considered that, as the renal pelvis probably contained non-opaque stones, a capacity of twenty-five cubic centimetres indicated hydronephrosis.

Calcification in an Adenoma of the Thyreoid.

Dr. King's last patient was a female, who, for many years had had an enlarged thyreoid with "lumps in it." At the age of forty she had manifested symptoms of hyperthyreoidism which had subsided three years previously, concomitant with the development of hard masses in the gland. At the time of presentation there were no signs of hyperthyreoidism, but the heart was enlarged, there were signs of mitral regurgitation and she suffered at times from shortness of breath and œdema of the ankles. The thyreoid gland was very enlarged and contained a number of adenomatous nodules. In the right lobe there was a large, stony, hard mass. A skiagram revealed extensive areas of calcification in the gland.

Syphilitic Aneurysm with Dysphagia Probably Due to a Malignant Stricture of the Œsophagus.

DR. F. APPERLY showed a male patient, aged fifty-seven years. He had had a chancre thirty-five years previously and had been a fairly heavy drinker. For the past twelve months he had had some shortness of breath. In the last two months he had lost eight kilograms (seventeen pounds) in weight. His appetite had been very poor, especially for meat. The bowels had been open only when medicine was taken. For the past fortnight he had complained of vomiting, coming on immediately after food and in the last week after fluids also.

On examination the cardiac apex was in the sixth intercostal space ten centimetres from the mid-line. At the aortic area the second sound was accentuated and reduplicated and there was a soft systolic murmur. Suprasternal pulsation was present. In the left arm the systolic blood pressure was 120 and the diastolic 80 millimetres of mercury, while in the right the systolic pressure was 170 and the diastolic 90 millimetres of mercury. The lower border of the liver to percussion was ten centimetres below the costal margin. The blood had reacted strongly to the Wassermann test, although that of his wife had failed to react. A skiagram revealed a pulsating aneurysmal dilatation of the aorta pressing on the œsophagus. X ray examination after the administration of a barium meal, had shown that the barium was held up at the site of the aneurysm. Above this there had been reverse peristalsis and slight dilatation and below a very narrow stream. Dr. Baxter had passed an œsophagoscope and twenty-six centimetres from the teeth had observed a well-marked stricture bleeding easily and with some ulceration. He had regarded it as being probably malignant.

Leucoplakia.

Dr. Apperly also showed a male, aged sixty-three years. He had had gonorrhœa many years previously and thirty years previously had been treated for a stricture of the urethra. He had been a heavy drinker, but had smoked very little. His wife had had no miscarriages. Seventeen years ago he noticed a white patch on the left side of the tip of the tongue. This had gradually spread until it had covered practically the whole of the anterior half of the dorsum. It had never given him the slightest pain or discomfort. A blood test had been done many years previously and no reaction had been obtained. For the past three months following the extraction of several teeth he had complained of pain in the mid-epigastrium, coming on only at night and always relieved by lying "on his stomach." The bowels had been constipated. He had lost a little weight. There had been slight shortness of breath on exertion. He had been passing urine two or three times during the night. There had been no gastric symptoms except for slight fullness after meals.

On examination the tongue had manifested slight tremor and had been coated with leucoplakia over nearly the whole anterior half of the dorsum. There had been no tender-

ness, nor could any lumps be felt. The cardiac apex had been in the fifth intercostal space ten centimetres from the mid-line. The first sound at the apex had been muffled and the second sound at the aorta accentuated. There had been occasional "dropped" beats. The systolic blood pressure had been 180 and the diastolic 110 millimetres of mercury. The liver dulness had extended two fingers' breadth below the costal margin. In the left epigastrium there had been slight tenderness to deep pressure. The urine had a specific gravity of 1015, was acid and contained no albumin or sugar. No abnormal neurological signs had been detected. The Wassermann test had given the following results: by the Harrison method no response with the warm, but "++++" with the ice-box method; by the Bordet method no response with the warm, but "++++" with the ice-box method; by the Kolmer method, no response. The test had been repeated a week later and had given the same results except for a slight partial reaction to Harrison's warm method. On May 13, 1927, the administration three times a day of *liquor hydrargyri perchloridi* four cubic centimetres (one fluid drachm) and potassium iodide sixty centigrammes (ten grains) had been commenced. He had also been placed on a milk diet for a fortnight and he had continued this of his own volition with the addition of white meats. At the time of presentation the tongue had already commenced clearing at the edges and the patch of leucoplakia appeared to be less opaque in the centre. His general condition had much improved and the tongue tremor was disappearing.

Skiagrams.

DR. H. M. HEWLETT showed a number of interesting X ray films.

One film demonstrated a very large single cyst at the posterior part of the base of the left lung and displacing the heart forwards. Stereoscopic and lateral films, taken from a child, aged three years, showed three cysts about the size of small oranges in the right lung, a large cyst at the base of the left lung and a small ruptured cyst in the left upper lobe.

Another film was that of an example of achalasia of the œsophageal sphincter, the œsophagus being twelve centimetres in transverse diameter.

Films of the abdomen were also shown illustrating hydramnios with a four months' fœtus and a large ovarian cyst, this film being made for the differential diagnosis from pregnancy, but there were no fœtal parts.

A skiagram of an unusual fracture of the hyoid bone was also exhibited and films of an atypical case of hydropneumothorax, there being very little collapse of the upper part of the lung and the level of the fluid being in two steps, evidently due to a large number of adhesions.

An interesting chondrodystrophy was also exhibited, the X ray appearances not corresponding to any of the recognized forms.

Coloboma of the Macula.

DR. FRANK DAVIES presented a patient illustrating the ophthalmoscopic appearances of the very unusual condition of coloboma of the macula. In that region there was a well defined circular patch white in colour and of nearly the same diameter as the optic disc. Both the chorioid and retina were completely absent and one or two small vessels could be seen encroaching on it from the margin. Dr. Davies said that the condition was probably due to a congenital malformation.

Changes in the Ocular Fundus.

Dr. Davies also showed patients illustrating the ophthalmoscopic appearances of embolism of the central artery of the retina and central chorioiditis.

Iritis and Keratitis.

DR. FRANK SPRING showed a patient suffering from old iritis and keratitis in the left eye. Two years previously this had been supposed to be due to a septic infection in the region of the knee. Vision had been reduced to bare perception of light. There were now massive hæmorrhages into the vitreous. Dr. Spring regarded the condition as being probably tuberculous in origin.

Papillœdema.

Dr. Spring's next patient showed papillœdema with general swelling of the disc and retina, but with no exudates or hæmorrhages. The causation had not yet been investigated.

Xerosis of the Cornea.

Dr. Spring also showed a patient suffering from xerosis of the cornea, following on an old trachoma. The xerosis had rapidly cleared up after tarsorraphy had been performed, but when the lid margins were free it had recurred.

Retrobulbar Neuritis.

Dr. Spring also presented a patient suffering from retrobulbar neuritis, following on an infection of the ethmoid air cells. After the latter had been drained the vision had remained at $^6/_{60}$ and after antisyphilitic treatment, even although the blood had failed to react to the Wassermann test, the vision had rapidly improved and at the time of presentation was $^6/_5$ and $^6/_6$ respectively.

Congenital Changes in the Optic Disc.

Dr. Spring's last patient showed optic discs which appeared to be typical of glaucoma. The fields of vision were not contracted and the blind spots with an Elliot scotometer were only slightly enlarged. He regarded the condition as being congenital in origin.

Asthma.

DR. FRANK STONE showed a female, aged thirty-two years, who had been suffering from asthma and nasal catarrh for the past two years. In the last six months the attacks had become extremely severe, occurring every night and allowing only about two hours of rest. She had been losing weight steadily and her condition was becoming critical. Examination of the nose had shown diseased mucosa in the ethmoidal region with numerous small polypi. The tonsils had been septic. Both maxillary antra had been washed out and the returning fluid had been clear in each case. X ray examination had confirmed the diagnosis of ethmoidal disease and had indicated polypoid mucosa in both antra. Under general anæsthesia the anterior and posterior ethmoidal cells on both sides had been removed and both sphenoidal sinuses opened. Tonsillectomy had been performed and a radical operation done on both maxillary antra, in both of which gross polypoid change had been present with abscesses in the substance of the mucous membrane, but no free pus in the cavity. The sphenoidal sinuses had contained yellow pus and the ethmoids diseased polypoid mucous membrane. There had been a severe attack of asthma on the night following the operation. Since the operation three months previously she had been completely free of asthma, had put on 7·2 kilograms (sixteen pounds) in weight and had lost a chronic cough of many years' standing.

Dr. Stone's second patient was a female, aged fifty-five years, who had asthma for twenty years. She had had no nasal symptoms and came to hospital as a result of a routine examination of the nose by her doctor. Examination of the nose had revealed a large mass of polypi entirely filling the upper half of the nose and polypoid disease of the maxillary antra. Under general anæsthesia the anterior and posterior air cells on both sides had been removed and a radical operation done on both maxillary antra which had been affected by gross polypoid disease. Since operation four months previously she had had no asthma, had put on weight and had felt much better. Dr. Stone pointed out that the presence of such gross nasal disease without any symptoms, showed the value of nasal examination in all cases of asthma.

Diseases of the Accessory Sinuses of the Nose.

Dr. Stone also presented a female, aged twenty-eight years, who had been complaining for many years of pain behind the eyes, vertical headaches, very severe depression

and stuffiness of the nose. There had been no other symptoms. Examination had revealed numerous small polypi and diseased mucosa under both middle turbinates. Investigation had been otherwise negative. Under general anæsthesia the septum had been resected to allow of access to the ethmoidal cells which had been removed without the removal of any of the middle turbinates. The nose at the time of the meeting had a normal appearance, as the middle turbinates covered the field of operation. Since operation eight months previously the patient had been completely relieved of headaches, depression and eye pain.

Dr. Stone's last patient was a boy, aged six years, who eight months previously had had his tonsils and adenoids removed. His mother stated that he still had nasal catarrh and was dull and inattentive. Examination of the nose by mirror and speculum on two occasions had revealed a normal appearance. The maxillary antra had been washed out and were found to be full of pus; since then he had improved considerably. Dr. Stone said that he proposed to wash out the antra several times and if this did not effect a cure, intranasal antrum drainage would be established. He pointed out that children with persistence of symptoms after removal of tonsils and adenoids, should have the nasal sinuses investigated.

ANNUAL MEETING.

THE ANNUAL MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Darling Building, University of Adelaide, on June 30, 1927, DR. H. H. E. RUSSELL, the President, in the chair.

Annual Report of the Council.

DR. H. H. E. RUSSELL presented the Annual Report of the Council.

Annual Report of the Council for the Year Ended June 30, 1927.

Election.

At the Annual Meeting, held last May, the following were elected:

President: Dr. H. H. E. Russell.

Vice-Presidents: Dr. R. H. Pulleine and Dr. F. J. Douglas.

Honorary Medical Secretary: Dr. F. N. Le Messurier.

Honorary Treasurer: Dr. W. A. Verco.

Members of Council: Dr. G. H. B. Black, Dr. M. W. Sprod, Dr. A. K. Gault and Dr. R. E. Magarey. Dr. Poole and Dr. E.. A. Brummitt remained for another year.

Federal Committee Delegates: Dr. H. S. Newland and Dr. Bronte Smeaton.

Library Committee: Dr. A. A. Lendon, Dr. H. S. Newland, Dr. F. H. Beare, Dr. W. Ray and Dr. F. N. Le Messurier.

At the first Council Meeting, held in June, 1926, the following Subcommittees were appointed:

Scientific: The President and Dr. Dr. H. S. Newland, Dr. W. A. Verco, Dr. G. H. Black and Dr. F. N. Le Messurier (Convener).

Lodge and Ethical: The President and Dr. F. St. J. Poole, Dr. E. A. Brummit, Dr. M. W. Sprod and Dr. A. K. Gault, The Lay Secretary (Convener).

Post-Graduate Subcommittee: Dr. C. T. Ch. de Crespigny, Dr. H. S. Newland, Dr. W. A. Verco and Dr. F. N. Le Messurier.

Meetings.

Nine meetings were held during the year, one of these taking the form of a combined meeting with members of the Dental Society and another being a clinical evening at the Adelaide Hospital.

On the whole, the attendances were somewhat disappointing and it is hoped that members will show increased interest during the ensuing year.

The following papers were read:

1926—July:
- Dr. F. S. Hone, "Prevention of Tuberculosis."
- Dr. H. W. Wunderly: "Specific Treatment of Pulmonary Tuberculosis."
- Dr. H. S. Newland: "Some Surgical Aspects of Tuberculosis."

August:
- Dr. W. Sangster: "Tonsils and Adenoids in Children."
- Dr. E. A. Matison: "Middle Ear Infection in Children."

September:
- Dr. A. A. Lendon: "Medico-Legal Cases."
- Dr. A. F. Lynch: "Medico-Legal Experiences."

October:
- Professor J. B. Cleland: "Pathological Heart Specimens."
- Dr. A. A. Lendon: "Heart Disease from Life Assurance Aspect."
- Dr. E. F. Gartrell: "The Interpretation and Clinical Significance of Certain Electro-Cardiograms."
- Professor C. S. Hicks: "Digitalis Compounds."

November:
- Dr. Leonard Trott: "Factors Influencing Extraction of Teeth."
- Dr. L. C. E. Lindon: "Dental Extractions in Relation to Surgery."
- Mr. A. P. R. Moore: "Factors Influencing Extraction of Teeth."
- Dr. Guy Lendon: "Dental Extractions in Relation to Medicine."

1927—February:
- Clinical evening at Adelaide Hospital.

March:
- Dr. W. C. T. Upton: "Some Observations on Certain Therapeutic Agents in Dermatological Practice."
- Professor J. B. Cleland: (1) "The Pathological Lesions Present in One Thousand Consecutive Autopsies at the Adelaide Hospital"; (2) "Carcinoma of the Stomach, Gastric Ulcers and Duodenal Ulcers in One Thousand Consecutive Autopsies at the Adelaide Hospital"; (3) (*a*) with Dr. J. G. Sleeman: "Gas Infections of the Uterus with Jaundice Following Abortions"; (*b*) with Dr. F. H. Beare: "Case of Gas Infection of the Uterus with Jaundice Following Abortion; (*c*) with Dr. Malcolm Scott: "Multiple Scarring Ulcerations of the Small Intestines with a Similar Scar in the Duodenum"; (*d*) "Three Cases of Purulent Infiltration of the Thyreoid Gland"; (*e*) "Partial Rupture of the Uterus with Fatal Intraperitoneal Hæmorrhage"; (*f*) with Dr. J. G. Sleeman: "Pseudomyxoma Peritonei of Appendiceal Origin and a Case of Huge Mucocele of the Appendix."

April:
- Dr. B. H. Swift: "Professor Blair Bell's Lead Treatment of Malignancy."
- Dr. G. H. Burnell: "Some Urological Notes."

May:
- Listerian Oration: Dr. R. Marshall Allan, M.C., M.D., F.R.C.S (England): "The Future of Obstetrics."

Council.

The Council met on twelve occasions, the attendances being as follows:

Dr. H. H. E. Russell ..	12	Dr. E. A. Brummitt ..	10
Dr. A. K. Gault	12	Dr. H. S. Newland ..	9
Dr. B. Smeaton	11	Dr. W. A. Verco.. ..	9
Dr. R. E. Magarey.. ..	11	Dr. R. H. Pulleine ..	7
Dr. G. H. B. Black.. ..	11	Dr. M. W. Sprod ..	7
Dr. F. J. Douglas	11	Dr. F. St. J. Poole ..	6
Dr. F. N. Le Messurier..	10	Dr. C. T. Ch. de Crespigny	2

Library Committee.

The Library Committee met on one occasion, the members present being Dr. A. A. Lendon, Dr. H. S. Newland, Dr. W. Ray and Dr. F. N. Le Messurier.

Scientific Subcommittee.

The Scientific Subcommittee met three times, the attendances being: Dr. H. H. E. Russell 3, Dr. H. S. Newland 3, Dr. G. H. B. Black 3, Dr. F. N. Le Messurier 3, Dr. W. A. Verco 2.

Lodge and Ethical Subcommittee.

The Lodge and Ethical Subcommittee has had an exceedingly busy time, mainly due to the New Model Lodge Agreement. It held thirteen meetings, the attendances being: Dr. H. H. E. Russell 13, Dr. A. K. Gault 12, Dr. F. St. J. Poole 12, Dr. M. W. Sprod 11, Dr. E. A. Brummitt 8.

In addition, three special conferences were held, one with the South Australian Friendly Societies' Association and the other two with Dr. G. E. Jose and Dr. L. D. Cowling.

Post-Graduate Subcommittee.

The Post-Graduate Subcommittee met once, the members attending being: Dr. C. T. Ch. de Crespigny, Dr. H. S. Newland, Dr. W. A. Verco and Dr. F. N. Le Messurier.

Federal Committee.

Dr. H. S. Newland and Dr. Bronte Smeaton again represented the Branch on the Federal Committee and both attended the two meetings held in Sydney and Melbourne respectively.

London Representative of Branch.

Dr. T. P. Dunhill having represented the Branch in London on the Council of the Association for the maximum period of six years, Council approved of the suggestion of the Victorian Branch that this Branch should nominate Sir Jenner Verrall, LL.D., as the joint representative. Notification has now been received that Sir Jenner Verrall has been duly elected to represent the South Australian, Tasmanian, Victorian and Western Australian Branches for the year 1927-1928.

Representatives at Annual Meeting in England.

Dr. C. T. Ch. de Crespigny and Dr. Edgar Brown were nominated by Council as Delegates representing the Branch at the Annual Meeting of the Association held at Nottingham in July last.

Representation on Boards.

South Australian Dental Board: Dr. R. H. Pulleine and Dr. A. R. Southwood were nominated as the Medical Members on the Board.

Nurses' Registration Board of South Australia: Dr. F. Steele Scott was again nominated as the representative of the South Australian Branch of the British Medical Association.

Membership.

The membership of the Branch now stands at 409, being a net increase of 26 for the period under review. The number of new members elected was 23 and the names of three members were removed from the list on account of arrears of subscriptions. The balance represents the difference between transfers "in" and transfers "out," after deducting deaths.

It is with much regret that the deaths are recorded of Dr. C. T. Abbott, Dr. H. M. Evans, Dr. L. W. Hayward, Dr. F. S. Mathwin, Dr. W. B. Shanasy, Dr. M. J. Symons and Dr. A. C. R. Todd.

Listerian Oration.

This year the Council invited Dr. R. Marshall Allan, M.C., M.D., F.R.C.S. (Eng.), Melbourne, to deliver the Listerian Oration. Dr. Allan chose for his subject "The Future of Obstetrics" and ninety members were present. Council desires to thank him officially for his interesting address.

Post-Graduate Course.

It is with much regret that Council decided to allow the course to lapse this year, but in view of the exceedingly poor response, the labour and expense involved would not have been justified. However, it is hoped that greater enthusiasm will be displayed next year and that the advantages offered will be appreciated by a larger number of members.

Formation of Sections.

During the year Council issued printed Rules for Sections for special branches of medical knowledge and a copy was forwarded to every member. Printed annual report forms have also been prepared.

Council would appreciate a close adherence to the rules in question. If annual reports are furnished, as laid down, it will enable publicity to be given to the work accomplished.

One Section has already been formed, namely "Eye, Ear, Nose and Throat."

Model Lodge Agreement.

The amendment of the present form of Model Lodge Agreement, particularly with a view to securing a higher scale of fees without the supply of medicines, which is to be the subject of a separate agreement between the Friendly Societies and the Pharmaceutical Society of South Australia, Incorporated, has engaged the earnest attention of both Council and the Lodge and Ethical Subcommittee during the past twelve months. The new draft agreement has been discussed at a great number of meetings, some of which have been convened especially for the purpose, and Council has requested the present members of the Lodge and Ethical Subcommittee upon the expiry of the current term of office to act as a Special Lodge Subcommittee in order to carry the matter to completion.

At the Federal Committee meeting held in Sydney on August 25, 1926, a proposal was considered to devise and adopt uniform conditions of Friendly Society Lodge practice throughout Australia and it was agreed that the Federal Committee approved of the adoption of a Model Lodge Agreement for all the States based on the Common Form of Agreement in operation in New South Wales. Further consideration to be given to the matter after the Branches had had the opportunity of considering it and communicating their views to the Committee.

At a subsequent Council Meeting, held on September 2, 1926, appreciation was expressed at the preparation of the brief by the Lodge and Ethical Subcommittee and it was reported that it was being utilized as the basis for further consideration by all the other Branches.

The Lodge and Ethical Subcommittee continued to devote considerable time and thought to its original recommendations and a fresh brief was prepared for the Federal Delegates who attended the Federal Committee meeting held in Melbourne on April 27, 1927, a copy being also forwarded to each of the other Branches. Prior to this meeting a conference of lodge delegates from the various Branches was held with aview to drafting a Federal Model Lodge Agreement for consideration by the Federal Committee. Dr. H. S. Newland represented the Branch and the thanks of Council are due to him for his attendance. In the issue of THE MEDICAL JOURNAL OF AUSTRALIA, dated May 14, 1927, pages 730 and 731, will be found a report embodying the resolutions of the Federal Committee and Council is now only awaiting receipt of the approved draft form of Model Agreement from the Honorary Secretary of the Federal Committee before circularizing members. In the meantime Council has given six months' notice of termination of the existing Agreements with the Friendly Societies' Medical Association (Incorporated), commonly known as the Dispensary Lodges, and the Port Adelaide United Friendly Societies' Dispensary (Incorporated), both of which expire on September 30, 1927. In addition, a circular letter has been forwarded to every member, advising the necessity of giving three months' notice of termination of each agreement with each friendly society lodge in accordance with the provisions of Clause 1 of the present printed form of Model Lodge Agreement. This separate notice by lodge medical officers was necessary owing to the fact that the contract is an individual one between each medical officer and each lodge concerned. Council is hopeful that these protracted negotiations will

be concluded at an early date with material advantage to those members interested.

Certificates under Workmens' Compensation Acts.

In consequence of representations submitted to Council in regard to the printed instructions issued in connexion with certificates under Workmen's Compensation Acts, the whole matter received the most careful consideration and was then referred to the Solicitors of the Branch.

It was found that the printed slip in circulation represented incorrectly the legal position and that in actual practice the directions laid down were impracticable of observance in that the employers in many cases took up the position that they were not legally liable for payment of certificates. This attitude was upheld by legal opinion, but as the matter was one of such vital interest to many of the members, Council decided to obtain the opinion of King's counsel. This opinion fully endorsed the views of the Solicitors of the Branch, in consequence of which Council resolved that a new printed instruction be issued in lieu of the old one. A copy of the fresh printed slip, based on counsel's opinion, was forwarded to all members under cover of the Monthly Circular, dated March 24, 1927, and members are advised to see that the revised slip is pasted in the cover of the certificate book, both for ease of reference and also in order to obviate any misunderstanding that may arise.

Increased Lodge Benefits.

In accordance with the provisions of the *Friendly Societies' Amendment Act* passed last year, some of the friendly societies have amended their laws in order to provide for increased sick pay and funeral benefits.

It was found that existing lodge members desirous of contributing to the increased benefits and also new members joining under the higher scale, were being sent for reexamination or examination, as the case may be, to the lodge medical officers without anything to indicate that the examination was other than that provided for in Clause 2 of the present form of Model Lodge Agreement.

The matter was fully considered by Council and referred to the Lodge and Ethical Subcommittee which subsequently met delegates of the Grand Lodges of the Friendly Societies in conference. As a result of this, Council approved of the recommendations submitted by the Subcommittee, which were as follows:

1. Reexaminations.—The fee to be 7s. 6d. and to be paid by the examinee at the examination. A more detailed certificate than the present one to be used.
2. New Members.—Those applying for the increased benefits to be treated as in (1).
3. Unfinancial members again coming on the funds and applying for the new scale of benefits to be similarly treated, but the arrears of medical fees, received by the lodges and belonging to the medical officers, to be paid to the latter by all lodges and not only by some, as at present.
4. Certificate.—To be clearly marked "For Increased Benefits."

In approving of these recommendations, Council felt that the new provisions of the friendly societies were approximate to those of the insurance companies and that a more detailed certificate was necessary, justifying the payment of a higher examination fee.

These decisions were conveyed to the Secretary of the South Australian Friendly Societies' Association, and under the direction of Council members were advised that, pending finality being reached, they had the right to refuse to examine these cases, but Council recommended that a fee of 7s. 6d. be charged which should be collected from the examinee at the time of examination.

The present position is that the Friendly Societies' Association has referred the resolutions of Council to the various Grand Lodges for their consideration and report to the next quarterly meeting of their Association.

Contract Practices—West Coast.

Both Council and the Lodge and Ethical Subcommittee have devoted a very considerable amount of time during the year in an endeavour to clear up the state of affairs that has existed on the West Coast for a number of years. Council has consistently held the view that the system of contract practice, other than that provided for under the Model Lodge Agreement, is not only unfair to the medical practitioners concerned, but also precludes in many instances a satisfactory service being rendered to the inhabitants of the districts where this system is in vogue.

After prolonged action during which application for appointment to these positions has been the subject of a notice appearing under the heading of "Medical Appointments: Important Notice" in THE MEDICAL JOURNAL OF AUSTRALIA, Council has the satisfaction of being able to report that at the present time the West Coast is, as far as it known, entirely free from this objectionable form of agreement. The opportunity is taken of pointing out that Council will only sanction, apart from Friendly Society Lodge contracts, a system whereby a minimum annual remuneration is guaranteed to the medical practitioner by the inhabitants of a district where the conditions are such that a guarantee of this nature is considered reasonable in order to secure the services of a fully-qualified medical practitioner. Council desires to place on record its appreciation of the services rendered by Dr. W. A. Verco in this connexion.

Medical Clubs.

Several instances have been reported to Council of the existence of medical clubs which provide for the medical attendance of members and their families at a fixed annual fee, irrespective of any income limit. Council wishes to make it quite clear that it will not sanction this form of agreement, whether written or otherwise, and, before any such undertaking is entered into, members should submit full particulars of the proposal to Council for consideration. A careful perusal of the printed form of Model Lodge Agreement should serve as a useful guide to members.

Brass Plates.

On several occasions during the year Council has received reports having reference to the unethical action of some members in regard to their brass plates. Council therefore decided to issue the notice that appeared in the Monthly Circular dated April 21, 1927, and it is hoped that this will have the desired effect.

North-South Railway.

In view of the danger of an outbreak of epidemic disease, especially typhoid, in consequence of the congregation of a large number of men in connexion with the construction of the North-South Railway, Council forwarded representations to the Acting Federal Minister for Health.

Advices have now been received from the Commonwealth Director-General of Health to the effect that regulations, designed to prevent the occurrence of typhoid fever in the camps, have been promulgated and that a railway medical officer, responsible for the oversight of sanitation, is to be or already has been appointed.

Visit of Sir George Buchanan.

Sir George Buchanan, Senior Medical Officer of the British Ministry of Health, representing Great Britain, and Dr. Norman White, of the Health Organization of the League of Nations, attended the International Pacific Conference held in Melbourne last December. Council endeavoured to arrange for Sir George to address members of the Branch during his stay in Adelaide, but, unfortunately, he arrived on Sunday and proceeded to Melbourne that night, so that the original programme could not be adhered to. However, it was found possible to entertain both Sir George and Dr. White in the limited time available, the entertainment taking the form of an inspection of the Quarantine Station, per quarantine launch from the Outer Harbour, luncheon with the President and Council at the South Australian Hotel, followed by a motor run in the hills. By the kind invitation of Lady Stirling afternoon tea was partaken of at her residence and the thanks of Council are due to her for her hospitality.

Library.

Acting upon the recommendation of the Library Committee, Council decided that it was inexpedient at this stage for the Branch to endeavour to maintain a medical library. It was felt that, rather than have two incomplete libraries, it would be better to support the Darling Library at the University of Adelaide and to continue the arrangement whereby the Branch makes an annual contribution to the Library funds.

Council therefore decided to offer the books at the Lister Hall to the Council of the University and these have been accepted on the understanding that, after the University has selected such as it desires to retain, the balance will be distributed to the best advantage, preferably to the Universities of Western Australia and Queensland.

A number of works of historical value have been retained and will be stored until such time as the Branch once more has a hall of its own.

Council desires to place on record its indebtedness to Dr. A. A. Lendon for the time devoted by him to cataloguing the books and also for his assistance in conducting the negotiations with the University authorities.

It is the desire of Council that city members should appreciate the facilities available at the Darling Library. Under the existing arrangement, they, as members of the Branch, are entitled to make use of the Library and also to take books out under certain conditions.

Permanent Home for the Branch.

During the period under review the Directors of the British Medical Hall Company, Limited, have sold British Medical Association House, Hindmarsh Square, at what is considered a very favourable figure. A temporary arrangement was entered into with the purchasers whereby the Directors were enabled to make the Lister Hall available for the meetings of the Branch for several months subsequent to the sale, but that has now been terminated.

However, Council has been able to make arrangements with the University authorities whereby accommodation will be available in the Darling Building until further notice.

As is probably known to most members, the Directors of the British Medical Hall Company, Limited, have purchased a valuable block of land on North Terrace with a view to erecting a building with provision for permanent accommodation for the Branch. Certain complications and negotiations for an alternative scheme have caused delay, but it is hoped that a satisfactory outcome will be arrived at in the near future.

Suggested Formation of Medical Association of Australia.

Dr. H. S. Newland has submitted a notice of motion in connexion with the formation of a Medical Association of Australia reading:

> That this Council takes the steps necessary to promote the formation of a Medical Association of Australia, of which the present Branches of the British Medical Association in Australia shall be Branches.

This motion was carried by a majority of Council and action will be taken to bring the matter before members at a special meeting of the Branch.

(Signed) H. H. E. RUSSELL, President.

Financial Statements.

DR. W. A. VERCO, the Honorary Treasurer, presented the Financial Statements for the year ended December 31, 1926. The Financial Statements are printed below.

Election of Office Bearers.

The following office bearers were elected for the ensuing year:

President: Dr. R. H. Pulleine.
Vice-President: Dr. H. Gilbert.
Honorary Medical Secretary: Dr. E. Britten Jones.
Honorary Treasurer: Dr. W. A. Verco.
Members of Council: Dr. E. C. Wilson, Dr. P. T. S. Cherry.
Delegates to the Federal Committee: Dr. H. S. Newland, Dr. Bronte Smeaton.

Income and Expenditure Account for the Period ended December 31, 1926.

EXPENDITURE.	£	s.	d.
British Medical Association—Subscription Account	429	0	9
Payment for THE MEDICAL JOURNAL OF AUSTRALIA	336	10	0
Hire of Lister Hall	24	3	0
Federal Delegates' Expenses	20	17	0
Federal Delegates' Capitation Grant	36	4	10
Salaries	313	9	3
Telephone	4	14	7
Advertising	1	19	0
Legal Expenses	6	16	6
Exchange	0	4	0
Postage and Telegrams	37	0	6
General Expenses	17	17	5
Stationery, Printing etc.	25	19	4
Duty Stamps	3	14	6
Subscription to Australasian Association for Advance of Science	3	0	0
Lister Medal Presented	1	14	2
Depreciation of Plant and Fittings	17	16	6
Transfer to General Fund	217	5	5
	£1,498	6	9

INCOME.	£	s.	d.
Subscriptions Received	1,263	15	0
Subscriptions Due and Unpaid	173	15	0
Interest and Dividends	41	0	0
Profit on Medical Certificate Books	0	13	9
Profit on Sale Commonwealth Diggers' Loan Bonds	19	3	0
	£1,498	6	9

General Fund Account.

	£	s.	d.
To Transfer to Library Fund Account (177½ City Members' Subscriptions at 10s. each)	88	15	0
„ Balance Carried Down	1,559	5	6
	£1,648	0	6

1925.	£	s.	d.
By Balance Brought Down	1,430	15	1
„ Income and Expenditure Account (Transfer of Surplus for Year)	217	5	5
	£1,648	0	6

Library Fund Account.

	£ s. d.		£ s. d.
To University Library Grant, Amount Paid and Due	50 0 0	By Balance Brought Down	259 18 10
„ Balance Carried Down	308 5 10	„ Savings Bank Interest	9 12 0
		„ Transfer from General Fund Account (being 177½ City Members' Subscriptions at 10s. each)	88 15 0
	£358 5 10		£358 5 10

Balance Sheet as at December 31, 1926.

LIABILITIES.	£ s. d.	ASSETS.	£ s. d.	£ s. d.
Medical Defence Association Fund	1 0 0	Plant and Fittings	178 5 5	
Medical Benevolent Association	2 10 0	Less Depreciation, 10%	17 16 6	
Subscriptions Paid in Advance	3 3 0			160 8 11
Dinner Account Surplus	3 7 2	Delineascope (Library Fund)		55 2 10
University of Adelaide	37 10 0	Commonwealth Loan 5%, 1948 (Face value, £400), Cost		403 12 0
Library Fund Account	308 5 10	Lister Medals and Dies on Hand		20 5 10
General Fund Account	1,559 5 6	Badges		3 18 7
		Savings Bank (Library Fund)		214 8 0
		British Medical Hall Co., Ltd., Loan Account		450 0 0
		Subscriptions Owing		178 0 0
		Stocks—		
		Hospital Report Forms	1 5 3	
		Medical Certificate Books	8 17 7	
				10 2 10
		Cash Balances—		
		Petty Cash in Hand	1 19 4	
		Cash in Hand	1 0 6	
		Cash at Bank	416 2 8	
				419 2 6
	£1,915 1 6			£1,915 1 6

Audited and found correct with Books and Vouchers produced.

C. W. L. MUECKE, F.I.C.A.

Adelaide,
June 21, 1927.

W. A. VERCO, Hon. Treasurer.
G. W. BENNETT, Lay Secretary.

Library Committee: Dr. A. A. Lendon, Dr. H. S. Newland, Dr. F. H. Beare, Dr. W. Ray, Dr. E. Britten Jones.

President's Address.

DR. H. H. E. RUSSELL then read his address (see page 210).

Induction of President.

DR. H. H. E. RUSSELL, the retiring President, then introduced the new President, Dr. R. H. Pulleine and expressed the hope that his term of office would be happy and successful.

DR. R. H. PULLEINE thanked the members for having elected him and said that he would do all in his power to further the interests of the Branch.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

Mills, Dorothy Isabel, M.B., Ch.M., 1926 (Univ. Sydney), The Sydney Sanitarium, Wahroonga.

Correspondence.

SPAHLINGER.

SIR: Two years ago I spent six months in Leysin and Montana, two well-known Swiss sanatorium villages. During that time I was in a position to gauge both the medical and lay opinion of Spahlinger. Your leading article of July 23, 1927, sums up the position admirably.

We with our more intimate knowledge of disease are apt to smile indulgently at the attitude of "the man in the street" towards the "quack" and the "herbalist." In some respects we are even more gullible and in none more so than with regard to tuberculosis. Side by side with the Hôtel Stephanni at Montana further down the Rhone Valley at Leysin and at Davos in German Switzerland, are established sanatoria and cliniques whose worth no one disputes. There is no mystery in their methods. Rollier at Leysin welcomes visitors with open arms. He is constantly on the lookout for disciples to spread his gospel. As far as he is concerned we are still enveloped in heathen darkness. I venture to state that in no other country in the world is there less attempt to cope with the problem of tuberculosis than there is in this. It is left to politicians and the press.

Whilst in Montana I met two Australians who had come there for the Spahlinger treatment! One of them told me that he had been sent as a test case after medical examination by a well-known association in Australia. As you pointed out in your article, he was a strange man in a strange land, he had no money for treatment, none of his home mail had reached him and he was obviously dying. A few weeks later he went to London and died there. Rather a grim joke the whole affair!

Instead of holding out alluring baits to charlatans we should bestir ourselves in the matter of modern, well-equipped sanatoria. Their position is not of paramount importance. We must also give the dependants of the unfortunate "*malades*" a thought. Lastly there are the

children, those who throng the outdoor clinics of our children's hospitals. Belgium has provided a special clinic for hers at Montana in a foreign country. We, with our wealth of fresh air and sunshine, are letting it go to waste.

It is but a truism to say that our attitude towards tubercle is of vital interest to Australia. With few exceptions it is met with "a light job in the country" or "a holiday in Queensland."

Yours, etc.,

K. J. O'DAY, M.B., B.S. (Melb.).

395, Barker's Road, Kew, Victoria.
July 24, 1927.

SAND FLY BITES.

SIR: Dr. Hill writes asking for an efficacious remedy for sand fly bites.

At Woy Woy last summer my baby was badly bitten on the legs and feet by sand flies. Peroxide of hydrogen, dabbing with the "blue" bag, applying damp cooking soda *et cetera* gave relief which was only temporary, and did not stop the child scratching the bites.

Then I tried Friar's balsam (*tinctura benzoini composita*) with excellent results, the bites dried up rapidly and there was no further scratching.

Yours, etc.,

GERTRUDE C. BUZZARD DUNLOP, M.B., B.S. (Melb.).

Strathfield, New South Wales.
August 2, 1927.

SIR: Dr. Hill asks for something to relieve sand fly bite. If he will paint each spot with "New Skin" the irritation will stop at once. I generally find that sand fly bites irritate first thing in the morning and that this goes on for four or five days; if, however, the spots are painted over night the "New Skin" causes a constriction of the blood vessels and often stops the bites from causing any further trouble.

Yours, etc.,

W. J. STEWART MCKAY.

227, Macquarie Street, Sydney.
August 3, 1927.

THE ST. LEGER WILLIS FUND.

SIR: For the information of those who contributed to the St. Leger Willis Fund, may I ask you to publish the following statement in THE MEDICAL JOURNAL OF AUSTRALIA. On the death of Dr. Willis £10 was sent by the Medical Benevolent Association in advance for Mrs. Willis's immediate use. In response to the appeal made in the journal the sum of £106 4s. 6d. was subsequently collected.

This amount, *minus* the £10 advanced and *plus* the accrued interest, £5 10s. 6d., now totals £101 15s. which sum has been forwarded to Mrs. Willis's trustee at her request.

Yours, etc.,

E. S. LITTLEJOHN,
Honorary Treasurer, Medical Benevolent Association of New South Wales.

B.M.A. Building,
30, Elizabeth Street, Sydney.
July 28, 1927.

Lectures.

MIND AND MATTER.

As announced in THE MEDICAL JOURNAL OF AUSTRALIA of July 9, 1927, Dr. C. I. McLaren, Lecturer in Neurology and Psychology at the Severance Medical College, Seoul, Korea, delivered a lecture entitled: "An Hypothesis Concerning the Relation of Body and Mind" at the Queen's Hall, Melbourne, on July 14, 1927. Dr. R. R. Stawell was in the chair.

DR. MCLAREN dealt with the dependence of the body on the mind and claimed that this was more than an inference; it was a fact of experience. After having discussed the evidence on broad biological lines, he proceeded to relate instances of the alteration of mind through poisons, injuries to the brain, glandular deficiencies and other causes. He then examined the mechanistic inference of the materialistic philosophy and endeavoured to prove that such an inference was neither inevitable nor actually untenable. He devoted his attention to the difficulties which presented themselves to the mind of the medically trained individual relative to the law of conservation of energy. He sought to show in the light of relativistic mathematics, of the conclusions of Clerk Maxwell and of a non-mathematical suggestion of his own that these difficulties were not insuperable. He illustrated his reasons for disbelief in the theory that nervous impulses were the essential cause of mentality by reciting a fairy tale of an "avivorous" prince and an amblyopic princess who could not see the birds. He pleaded for a better teaching of mathematics by an increase of physical stimuli to the students. He professed himself a believer in interactionism along the lines of MacDougall's dualism, but sought to understand by the consideration of each how it was possible for the mind and the body to interact. He considered the nature of mind and then passed on to the consideration of the nature of matter, more particularly in the light of modern mathematical physics. He quoted the conclusion of his brother, the late S. B. McLaren, that matter was objectified thought. He held that such a conception could be related to the Hebrew-Hellenic culture out of which western civilization had grown.

In the concluding part of the lecture Dr. McLaren turned his attention to the cause of mental disease. He postulated that the assault upon the human "spirit," of which mental disease was a manifestation, was either indirect, of the nature of poisoning or injury to the brain, or direct of the nature of those things enumerated in the English Church litany: "From all blindness of heart, from pride, vainglory and hypocrisy, from envy, hatred and malice and all uncharitableness, from fornication and all other deadly sin and from all the deceits of the world, the flesh and the devil, Good Lord, deliver us!"

In recognizing the psychogenic origin of mental disease he admitted the partial truth of the Freudian theory, but held that it should be put in a perspective in which other psychic causes of mental ill-health were recognizable. He emphasized the necessity for and the importance of the differentiation between the indirect and the direct assault. He endeavoured especially to discriminate between the results that might legitimately be expected from physical treatment and those that might be expected from psychical treatment. He stated that the goal of physical treatment was a healthy bodily organ, be it brain or other organ, and nothing more. While general and adjuvant treatment should not be neglected, there was such a thing as specific treatment for specific conditions. The specific treatment for a fracture was rest of the limb with the fractured ends in apposition; the specific treatment for diphtheria was antitoxin. Removal of septic foci might hasten the healing of a fractured bone, but no one would propose to treat fractures by pulling teeth. He contended that the specific treatment for the psychoses and the psycho-neuroses was psychical or spiritual. The patients should receive such general care and treatment as was calculated to combat physical disease and to promote physical well-being, but this treatment would no more cure a psychosis than would the reestablishment of mere bodily health cure an ununited, overriding fracture. From psychic and spiritual treatment he looked for an integrated personality. He gave instances to illustrate his contentions. He professed his belief in prayer as one of the specific means of treating mental and spiritual maladies. He summed up his hypothesis in the following manner. It was an hypothesis which took cognizance of the facts of physiology and neurology. The facts were accepted, but he resisted the mechanistic inferences of a materialistic philosophy. The hypothesis, he claimed, related itself with the arresting conclusions which

the insight of modern mathematical physics had attained and at the same time was enlightened by the genius and inspiration of both Hebraic and Hellenic thought. It was a form of dualism, but he was not content to leave mind and matter in two diverse "universes of discourse." According to his hypothesis matter was seen as thought that had taken form. Further the organic world was visualized as thought that was taking living form. In the human being, the crown of a biological process in which the *élan vitale* of creative evolution had given birth to man, he recognized an organism fashioned by the same energy that had brought the animal world into being. Dr. McLaren said that by his hypothesis the human being appeared as a person in whom in a manner unique in Nature conscious and self-conscious thought was enshrined in living form. He explained that the analogy sometimes used in an attempt to figure the relationship between body and mind, that of a player and his instrument, fell short in the appreciation of the essential intimacy that actually existed. He would prefer to state that consciousness was the music of man's personality, that it was music at the heart of all things that made "the music of the spheres," that has also made potential a musical instrument, that made that instrument actual and that promised the reconstruction of a beautiful instrument. According to the hypothesis the body was the instrument by, through and in which men, who were "spirit," perceived meaning, held memory and achieved purpose. Body and mind were related. The λόγος had become flesh; the κόσμος was intelligible.

In the course of a short discussion DR. S. V. SEWELL emphasized the physiological basis of mentality. According to his view mind was a function of brain cells. He did not believe that human mentality had in it anything more permanent than that of the lower animals.

MR. D. K. PICKEN approached the subject from a mathematical and philosophical point of view. He recognized description and not explanation in physiological and evolutionary facts. He recognized explanation in "meaning" and in "spirit."

AN EXPEDITION TO THE GREAT BARRIER REEF.

CONSIDERABLE interest is being taken in an expedition of scientists, naturalists and anglers to the Great Barrier Reef. The expedition, of which a preliminary notice was published in our issue of May 28, 1927, is timed to leave Sydney on November 13, 1927, and Brisbane on the following day. Several of the coral islands at the southern end of the Great Barrier Reef off the coast of Queensland will be visited. Twelve islands will be explored. The date has been chosen to coincide with the breeding season of the sea birds and turtles. The observation of this forms the principal objective of the trip. The sea birds congregate in millions on the islands at this time. Angling for the big game fish that abound in the waters of the Reef, such as the leaping tuna, the coral cod and the kingfish, will provide a special attraction to many of the party. Even more sensational will be the sport among the huge sharks, rays and gropers.

The party will include ladies. It is proposed to establish camps on three of the islands in turn. The party will be away for one month. We have been informed that the first boat has already been filled and that there is room for a few more scientists and Nature lovers in the second boat. Application should be made without loss of time to the organizer and leader, Mr. E. F. Pollock, of the Royal Zoological Society of New South Wales and of the Royal Australasian Ornithologists' Union. His address is Carrington Avenue, Strathfield, near Sydney. Members of the expedition will pay the sum of forty pounds from Sydney and thirty pounds from Brisbane. This covers first class return railway fares between Sydney and Brisbane, sleeper accommodation on the outward journey and all expenses on the islands and boats. Medical practitioners who have taken up the study of ornithology or other branches of zoology, or who are keen fishermen, will find this expedition an ideal holiday. The two previous expeditions organized by Mr. Pollock have proved to be most successful and this one promises to be the best of all.

Public Health.

THE CONFERENCE ON INDUSTRIAL HYGIENE.

IN the report of the third conference on industrial hygiene held in Melbourne on May 26, 1927, the agenda paper containing nine items is set forth. The first item is the presentation of a report on the activities of the Industrial Hygiene Division of the Commonwealth Department of Health. The report is included. The Industrial Hygiene Division was established in 1921 for the purpose of stimulating and coordinating modern methods of preventive medicine for increasing the health, comfort and thereby the efficiency of the Australian workers. Educational measures are utilized to bring before the notice of employers, workers, medical practitioners and the general public the advantages that result from the application of the principles of industrial hygiene.

Occupational morbidity and mortality are being studied. The Commonwealth Statistician is analysing the amount of illness occurring among the five hundred thousand members of friendly societies. Next year an analysis of the illnesses and accidents among the employees of the New South Wales Government railways and tramways and of the Victorian and South Australian Government railways will be undertaken.

The Industrial Hygiene Division is acting as an information bureau for firms, trade unions, the medical profession and other bodies in connexion with the creation of industrial medical services. Much work of organization has been completed. In order that medical practitioners engaged in this work might conduct their duties on proper lines, the Federal Committee of the British Medical Association in Australia after consultation with the Division has formulated a model form of agreement between the industrial medical officer and the company or firm engaging his services (see THE MEDICAL JOURNAL OF AUSTRALIA, September 18, 1926, page 396).

All matters relating to the health of officers in the Commonwealth Public Service are referred to the Industrial Hygiene Division. Full-time medical officers at the General Post Office in Melbourne and in Sydney are required to examine applicants for admission to the service, officers reporting "sick" or "injured" on duty, to supervise all applications for "sick" leave and to maintain a strict surveillance over the working conditions. The trained nurses employed at the General Post Office in both cities also have prescribed duties.

The Industrial Hygiene Division assists State authorities and other bodies by undertaking field investigations into the health of groups of workers. For example, two hundred mine employees were examined radiologically at Bendigo in 1922; six hundred sandstone workers were examined clinically and radiologically in conjunction with the New South Wales Board of Trade in 1924. Various other investigations and inquiries have been carried out by the Division in cooperation with the individual States of the Commonwealth. It is pointed out that the Division is investigating with the Department of Physiology of the University of Sydney at the suggestion of the Australian Commonwealth Engineering Standards Association certain aspects of lead absorption among employees of the Broken Hill group of mines and among the smelters at Port Pirie. Some important work has been carried out in connexion with the *Commonwealth Navigation Act*.

(To be continued.)

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered under the provisions of *The Medical Act of* 1925, of Queensland, as duly qualified medical practitioners:

Goldsmid, Joseph Albert, M.B., 1895 (Univ. Sydney). Brisbane.

Goode, Arthur, M.B., B.S., 1894 (Univ. Adelaide), Cleveland.

Books Received.

A PRACTICAL TREATISE ON DISEASES OF THE SKIN FOR THE USE OF STUDENTS AND PRACTITIONERS, by Oliver S. Ormsby, M.D.; Third Edition, thoroughly revised; 1927. Philadelphia: Lea and Febiger. Royal 8vo., pp. 1277, with illustrations.

WHOLE-MEAL WITH PRACTICAL RECIPES, by Ettie A. Hornibrook; 1927. London: William Heinenmann (Medical Books), Limited. Demy 8vo., pp. 78. Price: 1s. 6d. net.

Diary for the Month.

AUG. 15.—New South Wales Branch, B.M.A.: Organization and Science Committee.
AUG. 16.—Tasmanian Branch, B.M.A.: Council.
AUG. 16.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
AUG. 17.—Western Australian Branch, B.M.A.: Branch.
AUG. 18.—Section of Medical Literature and History, New South Wales Branch, B.M.A.
AUG. 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
AUG. 23.—Illawarra Suburbs Medical Association, New South Wales.
AUG. 24.—Victorian Branch, B.M.A.: Council.
AUG. 25.—New South Wales Branch, B.M.A.: Branch.
AUG. 25.—South Australian Branch, B.M.A.: Branch.
AUG. 26.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. John McIntyre Eadie (B.M.A.) has been appointed Medical Officer to His Majesty's Gaol, Bendigo, Victoria.

* * * *

Dr. Leslie Stuart Kidd (B.M.A.) has been appointed Medical Officer to the Reformatory Prison, Castlemaine, Victoria.

* * * *

Dr. Kenneth Claud Purnell (B.M.A.) has been appointed Medical Officer to His Majesty's Gaol, Geelong, Victoria.

* * * *

Dr. Sydney Theodore Appleford (B.M.A.) has been appointed Medical Officer to the Reformatory Prison, French Island, Victoria.

* * * *

Dr. John Bell Ferguson has been appointed Medical Superintendent, Tuberculosis Division, Public Health Department, Victoria.

* * * *

Dr. Hugh Alton Chandos Wall (B.M.A.) has been appointed Government Medical Officer at Coraki, New South Wales.

* * * *

Dr. George William Parramore (B.M.A.) has been appointed Government Medical Officer at Holbrook, New South Wales.

* * * *

Dr. William Malcolm Sinclair (B.M.A.) has been appointed an Official Visitor to the Hospital for the Insane, Toowoomba, Queensland.

* * * *

Dr. Joseph Bernard Dawson (B.M.A.) has been appointed Honorary Assistant Gynæcologist at the Adelaide Hospital, Adelaide, South Australia.

* * * *

Dr. Rita Margaret McAnaney (B.M.A.) has been appointed Medical Inspector of Schools, South Australia, on probation.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xx.

ADELAIDE CHILDREN'S HOSPITAL, INCORPORATED: Honorary Physician to Out-Patients' Department.

THE QUEEN'S (MATERNITY) HOME, ROSE PARK, SOUTH AUSTRALIA: Resident House Surgeon.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to positions at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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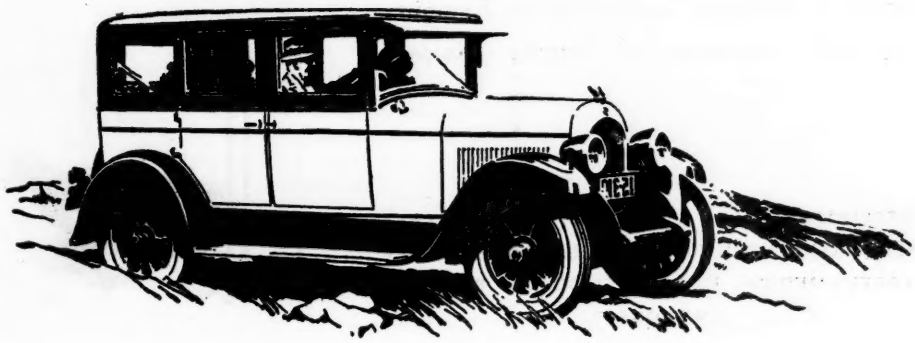

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